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H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

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EQUINE ENCEPHALOMYELITIS

Considering the horse situation throughout the United States at the present time, particularly in the agricultural districts of the West and Central West, an outbreak of equine encephalomyelitis of any extent this summer would be very unfortunate. It has been stated that practically all cases of the disease during recent years have been among farm horses, but this is not to be wondered at, considering that a very large percentage of the horses in the country at the present time are to be found on farms. On the other hand, even though there are in the neighborhood of 16,000,000 horses on the farms in the United States, many of these are virtually quarantined by reason of the fact that they rarely get to town nowadays, other means of transportation having largely replaced horses. The opportunities for picking up infections by coming in contact with other horses on such occasions would appear to have been reduced almost to a minimum.

Although considerable knowledge of a purely scientific character has been gained from the outbreaks of the disease which have occurred during the period 1930-1933, much remains to be known concerning it, particularly from an epizoölogical standpoint. Kelser has shown that at least one species of mosquito is capable of transmitting the filtrable virus of the disease and, even

though this form of transmission is not merely a mechanical one, the fact that encephalomyelitis has made its appearance in localities where this species of mosquito is not found suggests that there may be other vectors of the virus, either other species of mosquitoes or other insects, or that there may be some other fairly common means of transmission.

It has been pointed out that instances of contact infection are relatively rare. This fact complicates the situation and renders questionable the value of the prompt inauguration of the usual quarantine measures to prevent the spread of the infection from known infected animals to those that are exposed but apparently healthy. Practicing veterinarians should be particularly watchful of incipient cases of the disease, even though quarantine measures have not been demonstrated to be of any great assistance in controlling the infection. Exposed animals may be given a brief, temporary immunity through the agency of equine encephalomyelitis serum, although this is of questionable practical or economic value on a large scale. In this issue of the JOURNAL will be found a paper by Records and Vawter, dealing with a new method of actively immunizing horses against the disease. This is still in the experimental stage and its practical value remains to be demonstrated. However, the work already done is highly encouraging.

The control of encephalomyelitis appears to be complicated further as a result of the investigations which have been conducted in several laboratories during the past year on the nature of the filtrable virus. It appears that there are at least two strains of this virus operating in different parts of the United States. The so-called "western" strain of the virus appeared to be responsible for the severe outbreaks which occurred in Utah, Colorado, Nebraska and Kansas. The "eastern" strain appeared to be the cause of cases which appeared later in Virginia, Maryland, Delaware and New Jersey. Due to the fact that the eastern virus apparently differs in some immunological respects from the western type, this point must be given careful consideration in applying any method of biological prophylaxis.

Doctor Welch Dies

Dr. W. H. Welch, of Baltimore, Md., eminent pathologist and bacteriologist, died in Johns Hopkins Hospital, April 30, 1934, at the age of 84. He had been an honorary member of the A. V. M. A. since 1892.

DEAN GLOVER TO RETIRE

At the March meeting of the governing board of the Colorado Agricultural College, Dr. George H. Glover was appointed Dean of Veterinary Medicine for the period ending June 30, 1934, at which time he will retire from active service as head of the Veterinary Division. By way of explanation, it is pointed out that, although Dr. Glover has been the chairman and head of the Veterinary Division during the entire period of its existence, he has never had the title of dean. The heads of all the other veterinary colleges in the country enjoy this distinction.

Dean Glover will retire at the age of 70, exactly 50 years after he received his bachelor's degree from the institution with which his name has been identified for so many years. He studied veterinary medicine at Iowa State College and received his veterinary degree in 1886.

The course leading to a degree in veterinary medicine at the Fort Collins institution was started in 1906 and the first class was graduated in 1909. To date there have been 309 graduates in veterinary medicine and during the present college year there were 112 veterinary students enrolled.

Upon the retirement of Dr. Glover, June 30, Dr. I. E. Newsom, now Assistant Dean, will become Dean. He has been professor of veterinary pathology and head of the Department of Veterinary Pathology for a number of years.

EXECUTIVE BOARD ELECTIONS

Members of the A. V. M. A. located in Executive Board Districts 4 and 10 certainly have a wealth of material from which to choose their representatives on the Executive Board for the next five years. The usual thing happened in the primary elections held in both districts—there was a tie for fifth place on the ballot. In District 4 there was a double tie and in District 10 there was a triple tie, resulting in six and seven candidates being placed on the ballots in the respective districts. It is interesting to note that of the thirteen candidates in the two districts, seven are now or have been state veterinarians. Three college deans are among the candidates, as well as three pathologists. Each district has nominated an outstanding practitioner. Ballots have been mailed to all paid-up members in both districts. The polls will remain open until June 14.

District 4

BARNETTE, W. A. — Greenwood, S. C.

Practitioner. Graduate of Ohio State University, 1913. Joined A. V. M. A., 1915. Resident secretary for South Carolina, 1920-1921; member of Committee on Prevention of Transmissible Diseases of Animals, 1928-1929.

CARY, C. A. — Auburn, Ala.

Dean, College of Veterinary Medicine, Alabama Polytechnic Institute. Graduate of Iowa State College, 1885. Joined A. V. M. A., 1890. Fifth vice-president, 1907-1908; member of Executive Committee, 1910-1911; member of Committee on Reorganization, 1915-1916; chairman (1918-1919 and 1925-1928) of Committee on Resolutions; president 1919-1920; member of Committee on Revision of Constitution and By-laws, 1922-1924; member of Committee on Legislation, 1929-1934; member of Executive Board, District 4, 1929-.

DIMOCK, W. W. — Lexington, Ky.

Professor of Veterinary Science, College of Agriculture, University of Kentucky. Graduate of New York State Veterinary College, Cornell University, 1905. Joined A. V. M. A., 1906. Resident secretary for Iowa, 1910-1913; resident secretary for Kentucky, 1922-1924; member of Committee on Diseases, 1913-1915; member of Committee on Abortion, 1923-1924; chairman of Committee on Local Arrangements, 1925-1926; first vice-president, 1926-1927; member (1929-1931) and chairman (1927-1929 and 1931-1932) of Committee on Prevention of Transmissible Diseases of Animals.

KNAPP, J. V. — Tallahassee, Fla.

State Veterinarian of Florida. Graduate of Colorado Agricultural College, 1911. Joined A. V. M. A., 1926. Resident secretary for Florida, 1931-1933; secretary of Section on Sanitary Science and Food Hygiene, 1931-1933; member of Committee on Program, 1931-1933.

LEWIS, W. K. — Columbia, S. C.

State Veterinarian of South Carolina. Graduate of the Ontario Veterinary College, 1900, and of the McKillip Veterinary College, 1903. Joined A. V. M. A., 1915. Resident secretary for South Carolina, 1921-1922.

MOORE, WILLIAM — Raleigh, N. C.

State Veterinarian of North Carolina. Graduate of the U. S. College of Veterinary Surgeons, 1911. Joined A. V. M. A., 1926. Resident secretary for North Carolina, 1926-1933; member of Special Committee on Agricultural Extension Service, 1929-1931; member of Special Committee on Affiliation of State and Provincial Associations, 1929-1933; chairman of Committee on Resolutions, 1931-1932; member of Special Committee on Tuberculosis, 1933-.

District 10

BRUMLEY, O. V. — Columbus, Ohio

Dean, College of Veterinary Medicine, Ohio State University. Graduate of Ohio State University, 1897. Joined A. V. M. A., 1919. Member (1920-1921) and chairman (1926-1927) of Committee on Necrology; member (1924-1926 and 1931-) and chairman (1923-1924) of Committee on History; member of Committee on Schmidt Memorial, 1927-1928; member of Committee on Distemper, 1929-1931; member of Committee on Resolutions, 1930-1931; member of Executive Board, District 10, 1930-.

CLARK, C. H. Lansing, Mich.

State Veterinarian of Michigan. Graduate of Ontario Veterinary College, 1890. Joined A. V. M. A., 1916.

EDGINGTON, B. H.

Pathologist, Animal Disease Laboratory, Ohio Department of Agriculture. Graduate of Chicago Veterinary College, 1907, and of Ohio State University, 1912. Joined A. V. M. A., 1908. Resident secretary for Ohio, 1933-.

GILTNER, WARD East Lansing, Mich.

Dean, Division of Veterinary Medicine, Michigan State College. Graduate of New York State Veterinary College, Cornell University, 1906. Joined A. V. M. A., 1907. Representative to the National Research Council, 1924-1927; member of Committee on Resolutions, 1925-1926; member of Committee on Local Arrangements, 1928-1929; representative to the American Association for the Advancement of Science, 1928-.

HALLMAN, E. T. East Lansing, Mich.

Professor of Animal Pathology, Michigan State College. Graduate of Alabama Polytechnic Institute, 1910. Joined A. V. M. A., 1916. Resident secretary for Michigan, 1919-1921; chairman of Section on Education and Research, 1925-1926; member of Committee on Abortion, 1926-1930; chairman of Section on Research, 1930-1931; member of Special Committee on Agricultural Extension Service, 1933-.

HILTY, REUBEN Toledo, Ohio

Practitioner. Graduate of Ohio State University, 1907. Joined A. V. M. A., 1908. Resident secretary for Ohio, 1924-1925. President, 1927-1928; member of Committee on Education, 1928-1933; member of Special Committee on Affiliation of State and Provincial Associations, 1929-1932; chairman of Committee on Resolutions, 1933-.

KILLHAM, B. J. East Lansing, Mich.

Extension Specialist in Animal Diseases, Michigan State College. Graduate of McKillip Veterinary College, 1912. Joined A. V. M. A., 1917. Resident secretary for Michigan, 1921-1922, 1924-1927, 1931-1933; member of Committee on Local Arrangements, 1928-1929; member of Committee on Veterinary Biological Products, 1929-1930; member of Committee on Bang's Disease, 1930-.

STATE BOARD EXAMINATION

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. June 20-21, 1934. Application must be on file at Bureau 15 days prior to examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

Professor Götze has been elected rector of the Veterinary High School at Hannover, Germany.

Will Rogers says: "I love a dog. He does nothing for political reasons."

APPLICATIONS FOR MEMBERSHIP

(See January, 1934, JOURNAL)

FIRST LISTING

DEAN, BEN H. 5701-C Gifford Ave., Maywood, Calif.
D. V. M., Kansas State College, 1932
Vouchers: A. G. Feers, L. M. Hurt and R. R. Dykstra.

FANSLAU, CHARLES E. 120 Roseville Ave., Newark, N. J.
D. V. M., Cornell University, 1917
Vouchers: Earle B. Hopper and C. P. Zepp.

JOHNSON, M. J. Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1932
Vouchers: C. D. Lee and Geo. R. Fowler.

OMER, CHARLES R. 217 Church St., Rahway, N. J.
D. V. M., Kansas State College, 1929
Vouchers: I. Zimmerman and L. D. Ives.

SMITH, DWIGHT A. Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1932
Vouchers: C. H. Covault and C. D. Lee.

WHITLOCK, S. C. Iowa State College, Ames, Iowa
B. S., Michigan State College, 1927
D. V. M., Michigan State College, 1929
M. A., University of Michigan, 1931
Vouchers: H. L. Foust and C. D. Lee.

WICKER, H. E. 209 Welch Ave., Ames, Iowa
B. S., University of Wisconsin, 1929
D. V. M., Iowa State College, 1932
Vouchers: C. D. Lee and S. H. McNutt.

WORKMAN, TELFORD WM. Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1932
M. S., Iowa State College, 1933
Vouchers: Chas. Murray and S. H. McNutt.

Applications Pending

(See April, 1934, JOURNAL)

SECOND LISTING

Crane, D. B., 65 Grove St., Mount Kisco, N. Y.
Jensen, V. K., Woodlake, Minn.
Jerstad, A. C., c/o Puritan Poultry Corp., Atascadero, Calif.
Kilpatrick, W. C., 17 N. 15th Ave., Yakima, Wash.
Nisbett, E. E., 609 Broad St., Nashville, Tenn.
Rust, John Howard III, 23 Forest St., Wellesley Hills, Mass.
Walgren, O. E., Platte Center, Nebr.
Wright, Stuart L., 105 Bay St., Glens Falls, N. Y.

The amount which should accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1935, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Connecticut Veterinary Medical Association. Middletown, Conn. May 2, 1934. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. May 2, 1934. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. May 2, 1934. Dr. Harley B. Wood, Secretary, 2754 Meramec St., St. Louis, Mo.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. May 8, 1934. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

San Diego County Veterinary Medical Association. San Diego, Calif. May 8, 1934. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Hudson Valley Veterinary Medical Society. White Plains, N. Y. May 9, 1934. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. May 9, 1934. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. May 10, 1934. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Interstate Veterinary Medical Association. Elks Building, Omaha, Nebr. May 14, 1934. Dr. G. L. Taylor, Secretary, Plattsmouth, Nebr.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. May 15, 1934. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. May 16, 1934. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Keystone Veterinary Medical Association. Philadelphia, Pa. May 23, 1934. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Colorado Veterinary Medical Association. Fort Collins, Colo. May 24-25, 1934. Dr. J. C. Flint, Secretary, Colorado Agricultural College, Fort Collins, Colo.

Northeast Kansas Veterinary Society. Jayhawk Hotel, Topeka, Kan. May 31, 1934. Dr. E. H. Lenheim, Secretary, 326 City Building, Topeka, Kan.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. A. & M. College of Texas, College Station, Texas. June 4-8, 1934. Dr. D. Pearce, Secretary, Box 335, Leonard, Texas.

American Association of Medical Milk Commissions. Joint meeting with Certified Milk Producers' Association. Statler Hotel, Cleveland, Ohio. June 11-12, 1934. Dr. Harris Moak, Secretary, 360 Park Place, Brooklyn, N. Y.

Oklahoma Veterinary Medical Association. Southern Hotel, El Reno, Okla. June 11-12, 1934. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

California State Veterinary Medical Association. Joint meeting with American Association for the Advancement of Science. Berkeley, Calif. June 18-23, 1934. Dr. Geo. M. Simmons, Secretary, 1386 Golden Gate Ave., San Francisco, Calif.

Michigan State Veterinary Medical Association. East Lansing, Mich. June 26-27, 1934. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

Montana Veterinary Medical Association. Billings, Mont. June 28-29, 1934. Dr. Hadleigh Marsh, Secretary, Agricultural Experiment Station, Bozeman, Mont.

New York State Veterinary Medical Society. Rochester, N. Y. June 28-29, 1934. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. June 28-29, 1934. Dr. Lee M. Roderick, Secretary, North Dakota Agriculture College, State College Station, Fargo, N. Dak.

South Carolina Association of Veterinarians. Joint meeting with North Carolina State Veterinary Medical Association. Spartanburg, S. C. July 10-11, 1934. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

National Veterinary Medical Association of Great Britain and Ireland. Edinburgh, Scotland. July 30-Aug. 3, 1934. F. Knight, Esq., General Secretary, 2, Verulam Buildings, Gray's Inn, London, W. C. 1, England.

Poultry Science Association. A. & M. College of Texas, College Station, Texas. August 7-10, 1934. Prof. D. H. Reid, President, A. & M. College of Texas, College Station, Texas.

Twelfth International Veterinary Congress. Waldorf-Astoria Hotel, New York, N. Y. August 13-18, 1934. Dr. H. Preston Hoskins, General Secretary, 221 N. La Salle St., Chicago, Ill.

SOME OBSERVATIONS RELATIVE TO AILMENTS OF INMATES IN A ZOÖLOGICAL COLLECTION*

By J. A. CAMPBELL, *Toronto, Ont.*

EARLY DAYS OF THE ZOOS

In the early days, when establishing a zoölogical collection, the care and management of the exhibits, in most cases, were entrusted to men who had shown interest in some phase or other of wild life, such as animal-dealers or their employés, ex-circus men, hunters, trappers, taxidermists, or students of natural history. Such men possessed natural ability, were resourceful, and had a fondness for the work. Everything pertaining to the welfare of the specimens in their charge was left to their own discretion and the animals reflected the care they received. That was fine while the animals were well, but when cases of injury or sickness arose, then it was a different matter.

Lacking training in medical science, these men were scarcely fitted to deal with the sick and injured. Any knowledge they had in this regard was simply acquired from non-professional men and even when they were nonplussed, they were inclined to seek help from a similar source. Yet, they undertook to treat mammals and birds, with "worms" as their stock diagnosis. A zoo is a great place to receive suggestions on the treatment of sick animals. It is usually the mecca of those who have been in contact with wild animals in some way or another, which seems to qualify them to be authorities on this subject.

When some particularly valuable or popular member of the collection became sick and the efforts of those in charge had failed to relieve the sufferer, someone would conceive the idea of calling a veterinarian, who generally arrived on the scene towards the last. His opportunity of effecting a cure was thus restricted. In this manner, the impression was created that professional services were not of much avail where wild animals were concerned. There were, perhaps, some grounds for this attitude, as many were not familiar with the diseases and treatment of animals other than domestic.

In recent years, however, a veterinarian's training prepares him for work in a wider sphere of usefulness. His knowledge of zoötechnics, laboratory procedures and methods of sanitation, as well as therapeutics, eminently qualifies him to safeguard and treat the inmates of a zoo. Those in charge of the administration of zoölogical institutions realize this, and the veterinarian

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

has now been given a freer hand and made responsible for the well-being of the animals to a greater extent.

Veterinary Surgeons' Use to Zoos

Fortunately for the veterinary profession, the men who have become interested in zoölogical work in this country have amply proven the great benefit a veterinarian can be to a collection of captive wild mammals and birds. Their accomplishments have been recognized, not only by zoölogists in this country, but all over the world.

Much of the credit for the great strides that have been made in this phase of our work is due to Dr. W. Reid Blair, of the New York Zoölogical Park. He was the first to demonstrate the importance of proper feeding, sanitation and natural surroundings in the health and appearance of captive animals, and with sympathetic understanding of their needs, he set to work to improve their condition.

Through the efforts of Dr. Blair, a hospital and quarantine quarters have been installed at the Bronx Zoölogical Park, New York, which have been a most important factor in controlling disease among the inmates. This hospital, the first of its kind, containing medical and surgical wards, operating-room, pharmacy and kitchen, quarantine ward and research laboratory, has been an inspiration to everyone interested in zoölogical collections.

I came in touch with Dr. Blair some twenty years ago, when he gave a lecture on "Diseases and Treatment of Captive Wild Animals" at the Ontario Veterinary College, and since that time I have found him a never-failing source of help when appealed to. A long-distance telephone call always elicits willing and helpful advice. About ten years ago, Dr. Blair suceeded Dr. Hornaday as Director of the New York Zoölogical Park, one of the most important positions in the field of science. This position keeps him so occupied that the duties of veterinarian have now been taken over by Dr. Charles V. Noback, who has made some very remarkable contributions on several phases of diseases peculiar to wild animals in confinement.

Other veterinarians who have become prominent in the zoölogical field are: Dr. Reuben Hilty, past president of the A. V. M. A., who became suddenly famous when he demonstrated the possibility of restraining a leopard by holding its tail; Dr. R. A. Kammerer, who is held in high esteem for the outstanding work he has performed at the Saint Louis zoölogical collection and Dr. F. A. Crandall, Delaware Park Zoo, Buffalo, N. Y., who has made a study of the care, management and diseases of elephants.

Clinical Diagnosis

Clinical diagnosis of captive wild mammals and birds is a great deal more difficult than it is with the domestic species with whose disease symptoms we are familiar. This knowledge has been acquired not only through years of observation and study, but we have the advantage of being able to handle our live stock. This allows a closer and more thorough physical examination without any objection to speak of on the part of the patients.

The physical examination of wild mammals and birds in confinement is handicapped by their pronounced natural instinct of self-preservation, and their inborn fear of mankind. Constantly alert, and suspicious of any unusual action, even on the part of those to whom they have become accustomed, the appearance of a stranger adds to their alarm. This fear is sure to be intensified, with the result that symptoms which may exist will be quite masked by agitation. Therefore, unless the subject is *in extremis*, a close clinical inspection should be made as unobtrusively as possible. Examination by the two important aids—pulse and temperature—are out of the question in most cases, as their use will not be permitted.

In a collection of wild mammals and birds, there are many representatives of the groups classified by zoölogists. A sick specimen may be a mammal, bird or reptile; diurnal or nocturnal; one of the Primates; Carnivora, Herbivora or Insectivora; an elepant, eagle, wombat, widgeon, kangaroo, kagus, lion or linnet. A zoo veterinarian may find himself treating a lion cub for tetany, a giraffe for tracheitis, or acting as obstetrician for an egg-bound sulfur-crested cockatoo.

Not only is there a great diversity of species and therefore of habits, but from what we know of our domestic live stock, each one must have its own peculiar characteristics and reactions relative to the manifestation of disease symptoms. Each of our domestic species exhibits symptoms entirely its own when suffering from a diseased condition that may be common to all.

Take for example a horse and a dog; both suffering from gastritis, due, we will say, to a toxic amount of arsenic consumed with the food. Their responses will be quite different. The horse will not vomit, but will manifest severe colicky pains; whereas, vomiting is a pronounced symptom with the dog and there are few, if any, signs of pain evinced, notwithstanding serious erosions of the gastric mucosa. How much more of a problem must it be, then, to interpret disease symptoms of mammals and birds that have only recently been subjected to confine-

ment, and are widely divergent in their food, habits and behavior.

By holding a coroner's inquest on all specimens, compiling details of every kind relative to history, housing, disease symptoms, treatment, postmortem examination, pathological and laboratory findings, and then backchecking on each with the keeper of the dead animal, is the only method by which we can hope to build up a scientific and rational basis for clinical treatment of captive wild mammals and birds, and at the same time create respect and confidence in veterinary services upon the part of owner and caretaker.

The incidence of disease and the occurrence of injuries are much more prevalent among fresh arrivals than among the older zoo denizens. The strangeness of new environment is very disturbing and disconcerting to all animal life. Worse than this, the majority of newcomers are not in good physical condition. Muscles are soft and wind poor; generally they are in a rundown condition, and in the case of birds, the plumage is sure to be more or less the worse for wear. Should they be turned into large enclosures without ample time to build themselves up and improve their muscular system and constitution, they are likely to encounter disaster or even sudden death from over exertion. Birds with damaged wing feathers that endeavor to fly may be severely injured by striking some object or fall heavily to the ground in their futile efforts. A new arrival usually becomes the object of curiosity on the part of the others with which it is placed; may meet with a hostile reception and, if unable to escape, hide or protect itself, is liable to be injured, perhaps killed, if not rescued in time.

Waterfowl that have not had the opportunity of keeping their feathers oiled, during the long journey in a confined space with lack of bathing facilities, will be at a great disadvantage if liberated among other birds in a pond. Indeed, with their muscles weak and plumage damaged and not waterproofed, they will be lucky to escape death by drowning while being pursued by some aggressive bird that resents the intrusion of the newcomer. Loss of wing feathers is also a serious matter and care must be taken to protect such individuals from exposure to cold, draughts or chills. The timid, fast-moving little ruminants are apt to get seriously hurt by colliding with fences when first liberated into their enclosures.

There is always the possibility that some contagion may have been picked up at an animal depot, or from an infected crate during the journey from the collecting depot in the native habi-

tat of the animals. The contagion appears to remain latent in the system or else a temporary or transient tolerance or resistance is acquired. When these subjects are liberated into larger cages, diseases seem to flare up with suddenness. This happens quite often with birds which are prone to a greater number of microbial diseases than mammals.

Apart from the danger of exotic disease being introduced, new arrivals have also been the means of conveying virulent diseases common to our domestic animals, such as canine distemper, infectious feline gastro-enteritis and hemorrhagic septicemia. Doctor Blair mentions, in his most interesting and instructive book, "In the Zoo," the case of a ferret which was the means of infecting coyotes and wolves with canine distemper. In the London Zoo, serious losses occurred as a result of the introduction of infectious gastro-enteritis among the smaller Felidae. Lions and tigers are not susceptible, but leopard cubs appear to have no resistance against this infection.

It is very necessary that new zoo arrivals be placed in quarantine for a period of from two to three weeks or longer before being exhibited among other species in the collection; not only from a disease point of view, but in order to give them an opportunity to build up constitutionally for their own protection. A close check-up should be made for parasites. For the entozoa, a fecal examination is necessary, especially with Carnivora and Canidae, which seem to be more prone to worm infestations, while external parasites, such as mange mites of all sorts, fleas, lice and ticks, must not be overlooked.

There is, of course, a great variation in the nervous disposition of all animals. Some will settle down and commence to eat regardless of where they are. In fact they are apt to eat and drink too excessively with disastrous results, particularly birds. Those of a shy and retiring nature may take some days before being reconciled to new conditions and surroundings.

With some newly caught specimens, captivity has a depressing mental effect, particularly those taken after the instinct of fear is activated. Psychic anorexia is the marked symptom. Unless this can be overcome as soon as possible, the refusal to eat and its attendant consequences will quickly cause death. Some are capable of enduring deprivation from food for protracted periods and appear to be in good health and none the worse for the lack of food. This has been noticed in the lower orders of the animal kingdom, it being a well-known fact that reptiles possess a far greater tenacity of life than the higher mammals. The length of time that animals can go without food varies considerably.

Mammals will last longer than birds. Of the latter, Carnivora can prolong a fast to a greater degree than the Herbivora, which may go for a week to ten days; whereas, the small Insectivora cannot survive much longer than a day or two without sustenance. Generally speaking, the smaller the specimen, the greater the mortality.

We received an adult male leopard which appeared to be in the best of health when he arrived, but he just would not eat although offered all the delicacies enjoyed by big cats. The anorexia continued for twelve days until we gave him the warm, steaming stomach and intestines of a calf. The aroma from the viscera was too much for him to resist. He walked over to the mass, smelled it, took a lick, and finally carried it to a corner and ate a good meal. This was continued for a day or so and then the usual food was given him without refusal.

It often happens that young animals refuse to eat their natural food and crave for something unsuitable. A three-month-old lion cub when brought to us would take only milk or sweetened farinaceous food. This cub had been captured in the wild, after the mother had been shot and, perforce, was brought up on the bottle, being particularly fond of condensed milk. It absolutely refused to touch meat of any kind although everything possible was tried. This animal had become emaciated, and had a greatly distended abdomen, with complete alopecia. One day a live chub about three inches long was thrown to it. The movement of the fish attracted the cub's attention. After playing with it for a while, he finally ate it. More fish were offered and these also were devoured. The next day a larger fish was stuffed with raw meat and eaten with relish. From that on, improvement was rapid. In a week or so, hair commenced to grow, and in a month he was in good condition and well coated with hair.

Common or Everyday Occurrences

There are a number of common or everyday occurrences among inmates of a zoo. Injuries are perhaps the most numerous, which is only to be expected considering the natural instincts and uncontrollable motions of all wild animals. Some zoölogical inmates have a remarkable resistance to rough handling and severe body injuries, while others will succumb from shock due to fright when quite gentle efforts are made to capture them. Injuries include fractures of every description and lacerations or punctured wounds inflicted by the teeth, beak and claws, or antlers and horns of cage or enclosure campanions. Neighbor-

ing inmates also will readily seize the opportunity of biting a foot or tail which protrudes into their quarters.

Self-inflicted sores are due to animals licking and scratching some part of themselves, a failing of the different wild dogs, hyenas and monkeys, which also have the habit of licking one another.

Flies may be the source of great trouble by creating sores. Due to the efforts of the animal to rid itself of the flies, it will scratch and rub the parts, causing extensive sore areas. Oil of tar and petrolatum seem to be about the best treatment, as the mixture sticks more firmly to or near the spot. The use of a "Queen Elizabeth" collar is very effective in preventing animals from licking certain regions of the body.

Long-legged birds such as cranes, storks, herons and secretary birds frequently have their legs fractured. The length and slimness of their legs, as compared to the weight of their bodies, makes it difficult to balance themselves, and this, combined with their nervousness, renders treatment very difficult. Unfortunately, most of the fractures become compound, and irreparable injury is caused to the soft tissues by the patient's efforts to progress while in a weakened and suffering condition, but in the event of a simple fracture, good results may be obtained.

I treated a secretary bird with a simple transverse fracture in the middle of the metatarsus, with good results. The injury was received while the bird was being transferred from a crate. The bird was caught before the bones broke through the skin and was given immediate attention. A firm, light splint was constructed enveloping the whole length of the metatarsus. After the leg was padded to sufficient thickness, a thin strip of wood such as binds our fruit-baskets, was put in place, secured by light gauze and well painted with several applications of carpenters' glue. The bird was placed in a large roomy cage and kept very quiet, as they are of a very restless disposition. In three weeks, the splint was removed and the fracture found to be solidly united, but it was another three weeks before it began to use the leg sufficiently to be let out.

In short-legged birds, fractures of the leg are treated easily and repair very rapidly, even when compound. In the treatment of such fractures in small aviary birds, a very serviceable splint can be made by using the quill part of a feather obtained from larger birds, held in place by binding with thread and covered with a coating of glue or flexible collodion. The patient should be isolated in a cage with a soft floor.

Wing fractures are a simple matter to treat. The affected wing is held to the body with bandages, secured either by tying the material or securing it in place with strips of sticking plaster. It is necessary to include the uninjured wing as well, as it affords a support or brace and at the same time controls the bird's movements. Where a fracture is compound and is likely to result in a drooping wing, or occurs with a bird that is a wader or species of waterfowl, I usually resort to amputation.

FIG. 1. Coyote with a simple fracture of the radius, showing the splint in position.

With the smaller hoofed animals, such as goats, sheep, deer and antelopes, simple fractures respond to treatment nicely. Treatment should be tried even when the break is compound and close to the extremity of the leg. After the bones have been set, light splints are applied. The animal is then placed in close quarters and kept as quiet as possible in order to discourage

movement which might disturb the dressings. A daily inspection is made to insure there is not excessive swelling or blue discoloration of the skin evidenced, in which case immediate attention must be given and the bandage loosened, or removed entirely if necessary.

Males among the Cervidae become very aggressive and dangerous during the mating season. They will often gore other animals with great ferocity; deep punctured wounds are inflicted in the thoracic or abdominal cavities. If the injury penetrates the chest, there is practically no hope, but there is a chance when the abdominal cavity receives a deep thrust. Dr. Hilty mentions the treating of a nilgai antelope that had a punctured wound through the abdominal wall from which a loop of intestine six to eight inches long protruded. After controlling the animal with chloral hydrate, the wound was cleansed, the intestine returned to its place, and the peritoneum and muscular walls sutured with catgut and the opening closed with silk. A girdle of wide adhesive tape was wound around the belly and an antiseptic pad placed over the wound. Tetanus antitoxin was administered and as no rise in temperature resulted the antiseptic pad was left in place for ten days and then removed. A perfect healing resulted, and the same animal has since given birth to, and reared, a pair of twins.

I had a similar experience with a barasingha deer that had been gored by a male Himalayan thar. A mass of intestine, the size of a cocoanut, protruded. We were not long in driving the animal into a shelter and then into a crate which was covered with a tarpaulin, and administering chloroform. In twenty minutes, the animal collapsed on the floor of the crate, its head was secured and ether given by the concentrated method. As soon as the anesthesia was completed, the animal was drawn out of the crate, placed on its back and the protruding bowel returned, after it was cleansed as well as was possible. The muscle was sutured with No. 5 chromic catgut, the skin closed with silk, and drainage provided for. For a time it seemed dull and listless, probably some peritonitis, but in about three weeks it was quite itself again.

Feet

The feet of mammals and birds are often in need of attention. Constant use on soft or smooth floors will cause feet of hoofed animals to grow out abnormally, resulting in deformity and lameness in a very short time, necessitating trimming of the excessive growth. This is a comparatively easy procedure with Bovidae,

which are cast like cattle, but with Equidae, such as the nervous zebra, the operation is much more difficult and hazardous, as they are very apt to struggle continually while under treatment.

The feet can be kept worn down to proper requirements by having the surface of the floor or ground covered to a great extent with sharp crushed stones.

The members of the cat family, particularly lions and tigers, are frequent sufferers from ingrowing claws, if they are not provided with a scratch tree on which to work their claws, or are fed on boneless meat. The animal has to be put into a shifting-cage and the infected foot secured with ropes or straps and the offending claw clipped off quickly with bone-cutters.

The feet of elephants need constant care and attention and should be inspected daily. Periodically the feet should be trimmed and the nails cut. The sole of the foot is rough, with irregular fissures running through the surface. Hard substances such as broken glass, small stones, pieces of wire, nails and tacks, are easily picked up. If not removed, they will cause irritation and lameness. If they penetrate deeply enough, they are liable to cause extensive suppuration. When a performing elephant experiences a wound in the foot, the animal seems to realize its seriousness and will hold the foot up until the wound is attended to.

The bird in the aviary may be troubled with an excessive growth of nails, causing discomfort or lameness; or an accumulation of dirt in the feet may set up an irritation. Long claws will impede the use of the feet, and may become caught, with the result that a dislocation or fracture takes place. Quite often birds get their toes and feet frozen, even up to the proximal end of the metatarsus. The affected parts do not swell, but a dry gangrene sets in.

Bumble-foot appears among certain birds, chiefly from standing on hard floors for long periods. I have noticed this condition among cranes, particularly the Asiatic. Treatment which consists of cutting open the swelling and removing the caseous mass that forms, is unsatisfactory, as it often takes months for the wound to heal and the bird has to be kept standing in a salt solution, in which some hay has been placed to provide a soft footing.

Mange mite infestation of the legs is seen, usually among pheasants, but may occur in other birds as well.

Diseases of the Alimentary Canal

Diseases of the digestive tract are quite prevalent. These are due largely to overfeeding, the consumption of unsuitable food,

and continually eating off contaminated floors or ground that is limited in area.

A good index as to the state of the bowels can be formed from the appearance of the feces, and an observant keeper will always keep a close check on this. There is a marked difference in the feces of various species of birds. Those of the flesh- and fish-eaters are white and chalky, and are expelled with considerable effort; while the stools of the grain- and seed-eaters are more or less firm and round. We must remember that with birds, the excreta from both the kidneys and bowels meet at the cloaca and the apparent looseness or hardness of the feces may be due to a kidney condition instead.

Dysentery is a very common ailment among all species of birds. It may be due to irritation caused by improper food or some specific organism. There is always more or less straining while passing the feces and the diarrheal discharges sometimes accumulate around the vent and block up the passage. The discharge of the bowels may be quite watery and sometimes streaked with blood. The opposite condition—constipation—may occur with certain birds such as pheasants and other grain- or seed-eaters, that are fed consistently on dry foods. Enemas in such cases are quite as beneficial as with mammals.

Colics as we see them in the horse may be provided by the elephant, which is subject to the common forms such as indigestion, spasmodic colic, intestinal flatulency and impactions. The elephant is a very restless animal, continually swaying its body, flapping its ears and swinging its trunk, but when sickness appears, all this stops. The animal keeps very still. In cases of colic, this period of dullness will be followed by a sudden onset of acute pains and then the big animal will show his symptoms in a big way. He will lie down and start to roll and grunt. Some become excitable and even maniacal with pain.

One Sunday night, I was called to see a seven-year-old elephant that showed all the symptoms of spasmodic colic. In the intervals when an attack of pain came on, she would rush aimlessly around her enclosure with her trunk elevated and her mouth open, dashing against the partition with great force and then slump down on the floor where she would lie struggling until the pain subsided. It was an easy matter to treat this animal with medicinal agents. We scooped out the centers of some small oranges and then stuffed them with Epsom salt to the amount of a pound and a half, to which half an ounce of powdered ginger was added. These crude capsules then were tossed into her open mouth and later we administered an ounce of chloral hydrate in

the same manner, and also gave enemas when the animal was down. Two grains of arecolin and one grain of strychnin were administered hypodermically as well. There was not much relief noticeable from the first dose of chloral, so another dose was given two hours after, which had the desired effect of quieting the animal. Relief was obtained for an hour or so, and then the attacks recurred with greater severity.

Next morning, we gave three pints of raw linseed oil, two grains of arecolin and a half-grain of strychnin, and more enemas. At noon, she had a very violent attack and collapsed. Her trunk became limp and respiration ceased. I stood on the body, placing one foot on the chest wall and the other on the abdominal wall and grasping a beam that was just within my reach, pulled myself up and let my whole weight down on the chest. This was done repeatedly, thus forming a method of artificial respiration. During this process three-fourths grain of strychnin was administered and to our relief breathing was resumed. For two hours the animal lay still. There was an occasional twinge of pain which finally subsided. The massaging may have been the means of moving something in the bowels. We left her alone for several hours and then attempted to get her up but without success, so we resorted to slings for 24 hours or so until she had regained her equilibrium. Six large decubitals developed, two on either side of the head and one on each hip.

On another occasion, a female elephant in her early forties devoured a large quantity of cabbage that had been touched with frost. In a short time, she was suffering from an attack of what appeared to be intestinal flatulence. As the animal was crazed with pain, it was dangerous to administer medicine by mouth so we confined our treatment to frequent and copious enemas and hypodermic medications, giving two grains of arecolin and one grain of strychnin. The injection was made where the skin was thin, *i. e.*, in the chest wall just behind the elbow. An hour after, griping became more pronounced and a quick cathartic action was manifested. At the end of three hours, there was no perceptible relief, so another injection of two grains of arecolin and a half-grain of strychnin was given, which in due course produced an increase of griping and catharsis. However, an hour or so later, the symptoms began to subside and there was no further evidence of pain. The animal remained dull and listless for two or three days before she resumed eating in a rational manner.

I have treated elephants, three or four years old, with colicky pains, or "cramps," as the caretakers call them, and they have

responded readily to alcoholic stimulants to which powdered ginger was added.

One would think that, owing to the immense size of the elephant, the amount of food it consumes, and the large stomach and intestines, medicinal dosage would be greatly increased over that used for the horse. In Col. Evans' book on "Diseases of the Elephant" he makes a comparison of the doses as outlined by Steel, Slymm, Gilchrist and himself. Take for instance nux vomica. Steel says ½ to 2 drams; Slymm, 1 to 6 drams; Gilchrist, ½ to 2 drams, and Evans ¼ to 1¼ dram. Also the following dosage: potassium bromid 4-6 drams, potassium nitrate ½ to 1 ounce, carbolic acid 2 drams (well diluted), sulfur 3 ounces, Epsom salt 8 to 10 ounces, bismuth subnitrate 3 drams, linseed oil 1 to 1½ pint. I have seen circus men give half a gallon without excessive purging.

It appears that in India, the native land of elephants, treatments are left almost entirely to the mahouts. These men employ a multiplicity of drugs to make up their formulae or mussauls. They are strongly averse to the administration of purgatives, but depend largely on stimulants and carminatives. Most of their prescriptions contain many ingredients which consist of such hot things as ginger, pepper, garlic, onions, chillies, mustard seed, coriander and caraway seed, etc. They are mixed with flour in boiled rice and fed in that manner to mask the taste as much as possible.

The camel, more than any other ruminant, seems to suffer from the diseases of the alimentary canal as seen in cattle, such as tympanites, impactions of the rumen, constipation and diarrhea. The treatment and dosage are the same as for cattle. A purgative of 1½ to 2½ pounds of magnesium sulfate will usually give results in twelve hours, with frequent evacuations for a day or two, after which the bowel movements will be less numerous and soft pellets will be passed. It generally takes a day or so for the feces to return to normal.

I once treated a grizzly bear with profuse hemorrhage of the bowels—a condition very similar to that seen in the dog. This animal was so depressed that it offered no objections to my entering its den. The head was elevated by passing a loop of rope around the upper jaw behind the canine teeth. Half an ounce of subgallate of bismuth mixed with approximately half a pint of honey was smeared on the back of the tongue with a flat piece of wood. At the same time a half-gallon of normal saline solution was administered subcutaneously. One dram of adrenalin chlorid solution was given intramuscularly as well. In two hours, there

was no change and the treatments were repeated. After another period of three hours, on examination I found there was such an improvement that it was decided to be unsafe to go into the cage. From then on, he made good progress and in two days was quite well again.

Respiratory Diseases

In looking over postmortem records for the past ten years, I find that respiratory diseases come close to those of the digestive tract in the order of frequency among the inmates of the Toronto Zoo, with birds as the chief sufferers. There seem to be many more specific diseases to which birds are susceptible than is the case with mammals. They are confined chiefly to the upper respiratory tract. Besides certain infectious diseases of a more or less mild character, there is the avian diphtheria and other more definite mold infestations.

Roup is a disease to which almost all birds are subject. Young, unseasoned birds seem to be more susceptible, although occasionally roup occurs among the older residents, and unless they can be isolated, it is far better to destroy the affected bird as soon as the condition appears.

With the bigger apes, such as the chimpanzee and orang-outang, bronchitis and bronchial pneumonia are common occurrences. I have seen all kinds of remedies used and have come to the conclusion that the less you do in the way of medicinal treatment, the better. Nursing and nourishment, with lots of liquid, give far better results. With regard to pneumonias; they are usually fatal, more so with birds than mammals.

Contagious Diseases

Contagious diseases are not so common among mammals as among birds. The latter are prone to a large number, certain of which affect one species and not another, even when in close proximity, or in the same enclosure, but the one disease common to all in a zoölogical collection is tuberculosis. Although hemorrhagic septicemia crops up on this continent, I have as yet, fortunately, had no experience with it.

A feature of tuberculosis is the remarkable distribution it has with respect to the organs affected. The most striking instance of this is the decided immunity of the avian lung to tuberculosis, compared with that of the mammal. We rarely find any lesions in the chest area of birds. They are almost exclusively abdominal, affecting the liver, spleen and intestines; whereas, with mammals, tuberculosis invariably occurs in the thoracic cavity, with the lung as a favorite site and serous membrane next. This remark-

able difference, however, applies to other diseases as well. We find the abdominal organs of birds are more susceptible to diseases, while with the mammals, the lungs are the weakest organs of the body and the most often affected.

The lungs of the bird differ from those of the mammal. They are relatively half the size and a great deal more solid and vascular, with very little power of expansion. They have, besides, the great auxiliary respiratory system, the air-sacs extending throughout the body. This difference may be sufficient to account for their greater powers of resistance to disease, but when the lungs of a bird are affected, the symptoms are very pronounced and usually fatal.

The very insidious nature of tuberculosis makes a diagnosis difficult until it has been established for some time, particularly with birds, where the disease is confined to the liver and spleen. These organs apparently can stand considerable pathological changes without any noticeable ill-effect on the condition and plumage of the bird. Where the lesions are set up in the bowels, however, symptoms of diarrhea and general unthriftiness are quite pronounced. Certain species of birds are more prone to it than others. The Carnivora and Insectivora are not nearly so susceptible as the Gallinaceii and the Psittacideii. With the mammals, it is more prevalent among the Primates and antelopes. Tuberculous lesions of the bones are rare in mammals and birds, although quite common in the human.

In a zoölogical collection, there are three factors conducive to the occurrence of tuberculosis: the survival of the unfit specimens, which would not have had the chance to live in the wild; the ease with which it may be spread, and insufficient supply of fresh air, sunshine and exercise. In nature, these factors do not exist but the congregating habits of mammals and birds permit the spread of the disease. It is difficult to estimate the occurrence of tuberculosis in the wild, as sick specimens are soon killed and the remains of those that die are very quickly disposed of by the many scavengers existing in the untamed world. Tuberculosis does exist, however, as a number of instances have occurred where wild birds shot by hunters have been found to be infected.

Dystokia

Dystokia is not a frequent occurrence among the animals in a zoölogical collection, as captivity seems to limit the breeding activities of most species, that is, to the extent of actually giving birth to young. The incidence of dystokia might be greater if more animals would conceive, as the conditions under which they

live tend to produce two of the important factors responsible for difficult birth that are common to our domestic species; *viz.*, poor physical condition with its weakened muscular tone, due to lack of exercise, and rickets, which is so prevalent among certain species in captivity. Malpresentation, of course, can happen with a member of any species.

When dystokia occurs, the sufferer must be caught as soon and as gently as possible, and those that are vicious and difficult to control or are multiparous, should be placed under an anesthetic before delivery is attempted. Uniparous animals that show some part of the fetus extruding may be restrained for the brief period it takes to effect delivery without recourse to anesthesia, but highly nervous and vicious animals such as monkeys should be given ether, when it is quite a simple matter to extract a fetus, the vaginal passage being short and the bony structure of the young very pliable, especially the legs.

Should dystokia be due to deformities of the pelvis, a hysterotomy must be resorted to without any delay. We have had two such cases: a West African civet and an Angora goat. Fortunately both patients made good recovery. In the former case, three live fetuses were removed and successfully raised on a cat. We did not care to risk the young with the mother, as the operation and the subsequent wound, coupled with her carnivorous physiology, made us afraid that she might devour her kittens. In the latter, the Angora goat, the kid was left with the dam and there was no trouble whatever in raising it.

With birds, we have the very serious complication of birth as manifested by "egg-binding," quite a common occurrence in all species, from the diminutive waxbill to the huge ostrich. It may be due to several different causes; a deficiency of sufficient lime to give the eggshell the necessary degree of hardness, the excessive size of an egg, want of tone of the oviduct, or too much adipose tissue surrounding the genital passage, the result of lack of exercise or advancing age, and the influence of cold weather.

The symptoms, as a rule, are quite obvious. Affected birds will be found acting strangely on a perch or on the floor, with feathers ruffled, unable to fly and hiding in a corner. This condition may be very sudden in its onset, and if no relief is obtained, death from exhaustion may result in a few hours. On the other hand, a bird may linger for several days with less noticeable symptoms. Occasionally a bird will die unexpectedly and the cause will be found to be egg-binding. On examination, the vent is seen to be swollen, with the visible mucous membrane showing various degrees of congestion from a dark red to a black

in color. The egg may be close to the surface or out of reach in the canal.

Where the mucosa has a black appearance it is an indication that considerable inflammatory action has become established and that the structures involved have lost their elasticity and moisture, so essential for the delivery of an egg. Should the egg happen to be soft-shelled, it is a decided handicap, as its pliable state does not provide the support necessary for the action of the sphincter muscles of the oviductor cloacae, rendering the bird's labor efforts unsuccessful. In such cases the prognosis is unfavorable.

The consensus of opinion indicates that applied heat is of the greatest importance in the treatment. The bird should be placed in a warm oven for half an hour or so, or the vent held over steam for five to ten minutes while, at the same time, a warm lubricant is introduced into the passage with a feather or fine camel-hair brush. The patient is then returned to its nest, or placed in a cage that is heated, and if no results are obtained in an hour or so, the heat and lubricating treatment should be repeated until the egg is laid, whereupon, the depressed bird promptly regains its normal condition and spirits.

The administration of a laxative such as castor or olive oil, in doses proportionate to the size of the bird, a minim or two for a canary, five to ten minims for a parrot or macaw, up to a tablespoonful for a bird the size of the domestic fowl, is of decided benefit, as the general tone of the structures involved is benefited and an increase of fluids to the parts takes place.

Some authorities advise forcible extraction of the egg by breaking it up. A great deal depends on the size of the bird. It may be possible in the larger species, where there is plenty of room, but as yet we have experienced no favorable results from this method. Fluid extract of ergot of rye has been recommended. I have not tried it, but intend to administer pituitrin in the next case that occurs, and if an affected bird is large enough, I may resort to operative measures.

Acclimatization

A general improvement in the care and management of zoölogical specimens has taken place in recent years. Most outstanding is the acclimatization of exotic mammals and birds from tropical to temperate climates. The old idea was that such animals must be confined in heated buildings when the weather became cool. They were tightly "shut in" at the close of summer and kept there until the warm weather came around again.

It has been my experience, that when these animals were placed in winter quarters, they were prone to sickness until accustomed to the new heated atmosphere, and toward spring, after a long winter indoors, their resistance seemed to drop to a low ebb and they became liable to disease. The warm weather had to be well settled before it was advisable to place them in their outside enclosures. They were like "hot-house plants" and not in condition to withstand any sudden drop in temperature, especially if cold rains occurred.

Mammals and birds in good condition can stand sudden falls in temperature without ill-effect. Cold weather has an exhilarating and invigorating effect, and acts like a tonic, as it were. In South Africa, there is a great variation in the temperature within a twelve-hour period. It may be excessively hot during the day, with frost at night. Generally speaking, my experience is that heat is more injurious than cold. We have had more losses from the former.

The pioneer in the movement of acclimatization was Carl Hagenbeck, who proved that it could be successfully carried out in his European collection, and his method is now generally followed. Some twenty years ago, we commenced to allow all our monkeys access to their outside cages in winter, in fact, encouraged them to go out by placing food there.

I always make it a point to check up on the animals' behavior during the severe weather. On a bright frosty day, most of them are outside enjoying themselves. I have seen our South American, African and Indian Primates out of doors with the thermometer registering ten below zero.

Since adopting this system, I do not remember having had to deal with any respiratory disease among our monkeys with the exception of two chimps and an orang-outang which were purchased from a travelling menagerie about four years ago. They were not in particularly good condition, with poor coats, so it was decided to keep them indoors that winter, with the result that all three had cold after cold, and the two chimps came down with bronchial pneumonia. The next winter they had free access to suitable outside enclosures, and since that time they have had only one or two mild colds. We have found this plan to be successful with other species, such as lions, Grevy's zebras, Indian water buffalo, Indian hump cattle, Indian antelope, ostrich, emus, and some species of cranes, parrots and cockatoos. Elephants are unable to stand extremes of either heat or cold, owing to the large hairless skin surface.

Not only do tropical animals which have been subjected to outside exposure during the winter enjoy better health and con-

dition, but there is a great deal more economy in their housing maintenance. If these specimens were housed as they were formerly, expensive buildings and costly heating-plants would be required. We endeavor to arrange their shelters so that the heat furnished by the larger animals will be sufficient for the smaller animals as well. Manure is allowed to accumulate, which aids in heating the buildings, without any noticeable ill-effect in health.

FIG. 2. Six-week-old Grevy's zebra, born in the Toronto Zoo, enjoying a walk with its mother, with the thermometer registering fifteen degrees of frost. This bears out the contention that animals properly acclimated enjoy spending some time in fresh air, even in very cold weather.

There is hardly a day that these animals do not go outside. Even in the coldest weather, they look forward to it and seem to enjoy being allowed to do so, to stretch their legs and breathe the fresh air, even if only for a brief period. When spring arrives, they are in fit condition to be in the open a great deal of the time and will suffer no ill-effect from any sudden drop in temperature or if cold rains occur; whereas, there is always more or less anxiety when animals that have been inside all winter are placed in their outside enclosures.

FEEDING

Feeding the inmates of a zoölogical collection has always been a big problem and many of the losses sustained are due to the

lack of something essential to the particular species. Since the discovery of those accessory food factors, vitamins and minerals, many of the difficulties have been overcome. Their use in the diet has been the means of saving and prolonging life, improving general health and increasing breeding activities with greater success.

Rickets and other nutritional or deficiency diseases have been of serious concern to those in charge of animals in captivity. The proneness of confined animals to rickets fortunately is being overcome by our better knowledge of food values and the importance of sunshine in promoting natural growth and bone development.

The different articles of food containing vitamins and minerals are cheap and easily obtained and have very little taste. A very essential kitchen equipment in a zoo in connection with dieting is the meat-cutter or mincing-machine. It provides a means of thoroughly mixing foods that are rich in essential articles of diet which the particular mammal or bird needs. For instance, we can include tomatoes, lettuce, cabbage, spinach, bran, milk powder, bone meal, alfalfa meal, orange-juice, yeast and cod-liver oil. All these can be worked into a mass and a balanced ration given in whatever proportion necessary to most specimens.

Restraint

The manner in which the inmates of a zoo are secured for the purpose of treatment, when sick or injured, is vastly different now from what it used to be. Formerly, it seemed that wild methods had to be used for wild animals. No consideration was shown for suffering during the catching process, or for what the after-effects would be in the way of the animal's reaction to strenuous handling. Lots of men, rope and iron bars were available to overpower a subject if it happened to be a large and dangerous one, and different kinds of nets for the smaller and weaker specimens. Nowadays, the method is a great deal more gentle and humane as possible.

Much depends upon whether the securing of the animals is urgent or not. If it is found necessary to catch a large, highly nervous and suspicious one without delay, there is sure to be difficulty, but if there is no immediate hurry, such an animal may be caught without being very much upset, by withholding its food and coaxing it into its den or shelter and then into a small crate or shifting-cage with some favorite item of food. When once confined in the small space, the procedure of further restraint can be carried on without much disturbance, or risk to

the animal, by the use of chloroform or ether poured on a large pad of absorbent cotton and placed in the crate. I generally start with chloroform, as its action is quicker and there is less excitement. There is always difficulty in estimating the exact amount required, so it is always advisable to have a good supply on hand. As the fumes take effect, efforts should be made to bring the anesthetic as close as possible to the nose of the animal, by tying about a quarter-pound pad of cotton wool, bound with cloth, on the end of a stick and saturating it, and holding it close to the nose of the patient. As soon as the animal is subdued to the extent that a more direct application can be made, ether is substituted for the completion of the anesthesia. It is then placed on the operating-table, or wherever necessary for the treatment required.

Chloral hydrate may be administered per rectum. Its action is very profound and the patient can be subdued by its influence and subjected to any of the operations of everyday occurrence. At one time I believed there were great possibilities for the use of morphin in controlling lions and tigers with as good effect as the domestic dog, with none of the maniacal reactions such as the house cat exhibits.

Some years ago, I gave an adult tiger four grains of morphin and, in half an hour, the animal was lying quite still and we were able to administer chloroform without any resistance. On another occasion I gave an old lioness five grains of morphin. In a few minutes vomiting occurred and, half an hour later, we were able to rope her without any resistance to speak of and cut off an ingrowing claw. It appeared to be such an ideal method of controlling these large and dangerous animals—just to inject the drug under the skin while they were rubbing their sides against the bars of the cage and as their backs were being scratched. But, the next two animals, a lioness and a tigress, each of which received four grains of morphin, reacted in a very unsatisfactory and disconcerting manner, so much so, that I will never attempt it again. The effect of morphin on wolves, coyotes and hyenas is quite good. Three grains will subdue any one of them sufficiently to be handled. I gave five grains to a year-old black bear without any noticeable response.

Use of Medicines

The use of medicines in the treatment of zoölogical inmates is a great deal more limited than with domestic live stock, for the

good reason that it is more difficult of administration. If there is any difficulty in administering medicines, they are worse than useless.

Wild animals have a very keen sense of smell and are of an extremely suspicious nature. One might imagine that it would be an easy matter to convey what appear to be almost tasteless and odorless drugs in its food to almost any animal when hungry, but such attempts are seldom successful, except to the unintelligent and gross-feeding species.

We have known of monkeys escaping and taking to trees and, in efforts to catch them, have placed what was considered large doses of narcotics, such as chloral hydrate or morphin, in some favorite food (condensed milk, molasses or fruit), but they seem to sense that something is wrong, and if they do take any, it is not nearly sufficient to place them under control. Other means than the use of drugs are more effective in capturing truant monkeys.

I administered morphin hypodermically to a Rhesus monkey weighing thirteen pounds and found that it was able to stand a grain for every pound of body weight before there was a profound action, and then it always had one eye open. I have heard of chloral hydrate being given in water to zebras with good results, after withholding water for some time, or getting them gradually used to some mixture that could be used to convey the drug. I have tried it with wolves, bears of different species, wild dogs, etc., but never yet with any satisfactory results. There are a number of useful drugs with little or no taste or odor that can be administered in the food or water. Also, a taste for cod-liver oil is soon acquired.

An interesting fact is that great virtue is placed on peppers, chillies, ginger, cinnamon, garlic and similar hot stuffs. They are recommended by authorities in the treatment of small cage birds, falcons, poultry, pheasants and elephants. It has been recorded that, in nature, mammals and birds will seek roots and seeds of such plants.

In the administration of medicines to birds, care has to be used with liquids or anything that will enter the larynx. Substances, no matter what they are, going down the wrong way seem to be quickly fatal to birds. The best plan is to use gelatin capsules, 2-minim capsules for small species and 2-ounce capsules for birds of the ostrich family.

Surgery

Most of the surgery in zoölogical work is in connection with injuries, treatment of wounds, abcesses and amputations following compound fractures.

Monkeys offer opportunities for operations. I have performed seven or eight ovariectomies. They are about on a par with dogs in this respect. The uterus of a baboon is very similar in

Fig. 3. Chacma baboon under the influence of anesthesia, commenced with chloroform and completed with ether, in order to have bandages removed five days after ovariectomy.

shape and size to the upper part of a man's thumb, with the ovaries lying close to it. The entrance to the abdominal cavity is made on the median line, and the lower part of the wound should be about one inch above the brim of the pubes. The muscle and skin are closed with chromic catgut. I always make it a practice to bandage them up securely. In most cases, they do not interfere with the dressing. This operation is indicated to eliminate the very large and unsightly swellings that appear in the perineal region at the estrual period. There has not been

any noticeable alteration of their disposition, nor do they become fat or inactive after the operation.

It is interesting to note that the monkeys from the New World seldom, if ever, come in estrum, but most all the Old World Primates go through a regular estrual cycle. The African baboons are very regular in this respect, and the swelling of the sexual skin will attain the size of a man's head with the color of a ripe tomato.

We are quite often called upon to prevent birds from flying. By amputating one wing at the wrist joint, this may be achieved. The operation should, of course, be performed while the birds are a few days or a week old, and is accomplished by using an ordinary pair of scissors. Hemorrhage can be overcome by the use of tincture of iron, but with adult birds, the operation should be

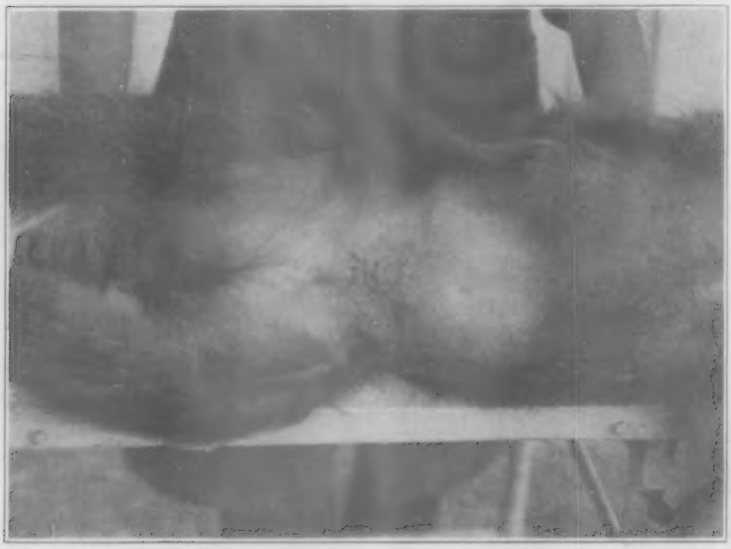

FIG. 4. Chacma baboon showing the wound after the bandage was removed, five days after ovariectomy.

done either under a local or general anesthetic, hemorrhage controlled and the wound bandaged. The bird should be kept quiet for a day or so until healing is established. Occasionally birds will pick and tear at the wound, causing a hemorrhage which may prove fatal.

I must have done this operation on at least one hundred waterfowl, such as swans, geese, ducks, cranes and herons, by saturating a piece of cotton batting in ether and holding it over the nostrils. In two or three minutes, the neck becomes limp, at

which time disarticulation at the wrist joint was performed, the blood-vessels ligatured and the skin flap sewn so as to cover the cut surface. The parts were painted with tincture of iodin, a bandage applied and left for twenty-four hours, and the birds kept quiet. Temporary pinioning may be done by cutting the long flight feathers of one wing; also, by tying the wings with tape, strips of leather or metal clips, they can be prevented from flying.

I have operated on several eagles and vultures for mycotic sinusitis, removing the fungus growth that collects and painting the cavities with iodin.

A very noticeable fact is that wounds of birds heal very rapidly without any evidence of pus formation to speak of. It is often necessary to operate on the crop of certain birds in order to evacuate the contents of the organ when it becomes impacted. This is done by removing the feathers from the affected area and making a perpendicular incision, drawing the skin to one side, entering the crop to one side of the skin opening with a similar incision, and removing the material. The wound is then sutured with fine catgut, and healing is completed within a few days, although restrictions have to be placed on the feeding for a short while.

In concluding, you will appreciate that I am not a zoo specialist, but simply a practicing veterinary surgeon who has been given an opportunity of acquiring a little knowledge of practice as it affects wild mammals and birds.

I have, therefore, offered you my observations with some diffidence, since I am speaking of what is in effect a hobby, without the pretense of seeming to appear an authority.

Discussion

Dr. S. W. Haigler: You mentioned the pinion operation in birds. What little experience I have had has been limited to Mallards and birds of that kind. The operation seems very simple and works very nicely in Mallards. It consists simply of removing a little section of the ligament.

A question I would like to ask is relative to intestinal parasites in the zoo. You did not mention them. Perhaps you are not troubled with them in the northern country.

Dr. Campbell: I should have mentioned that new animals should be quarantined and checked up. If we would make a habit of doing that, we would know more about them. We find that wolves are almost immune to intestinal parasites, while bears are prone to have them.

Dr. Haigler: It is my understanding that the Saint Louis zoo had considerable trouble years ago, and still has, controlling intestinal parasites.

Dr. Campbell: Fortunately we have fine facilities for research work and all I have to do is to send up a lot of bottles and we have specimens

from every species in the zoo. This last year we found that only lions and bears had any intestinal parasites.

DR. HAIGLER: What are the principal parasites? Any hookworm?

DR. CAMPBELL: No. A very nice thing is to check up. Go to the caretaker and tell him that you want to check up on the different species and it will impress him that you are sufficiently interested to do this. You will find, too, that you are attributing a lot of ailments to worms that are due to something else most probably.

MEMBER: I should like to ask Dr. Campbell about the period of gestation in an elephant. Undoubtedly there is a great deal of exaggeration in the stories we hear.

DR. CAMPBELL: There are a lot of wild tales told about the elephant. First of all, she comes in at around twelve years of age. The period of gestation varies from 20 to 22 months, and the elephant will breed every second year.

As I said, the period of gestation varies from 20 to 22 months, and it is said that when the period is extended to 22 months the offspring will be a male. The young are just the same as with the horse—they stand up and nurse shortly after birth. And they don't nurse with the trunk, the stories you hear to the contrary notwithstanding. They nurse with the mouth. The mammae on the female are right behind the forearm.

DR. E. L. QUITMAN: I should like to ask Dr. Campbell something in regard to the dosing of monkeys. Inasmuch as monkeys have some tricks in taking medicine that veterinarians should know, and we should have some tricks in order to circumvent the monkey tricks, I wish you would tell us what you can about them.

DR. CAMPBELL: My experience with monkeys is that they are just about as clever as can be; they have a very keen sense of smell. We once tried to fix up a banana with a grain or so of morphin, or something of that nature. The monkey the first thing smelled the banana and threw it down in disgust. It is a very difficult matter to get wild animals to take anything.

What do we do for a monkey? We give him calomel at times, but the best thing, in my experience, is to let the monkey alone. Keep him drinking. Give him something in the way of fluids; color it up a bit if necessary. Keep him busy and everybody is satisfied.

DR. QUITMAN: Isn't it true that you may administer a pill or tablet or capsule to a monkey, and although he may apparently swallow it and take it down into his stomach, in a moment or two he will throw it out?

The point I wish to bring out is this: The monkey, as you know, is a great thief and if you lay a capsule or tablet down within his reach, and just go away, as soon as your back is turned, he is apt to make a grab for that and down it goes.

That is my experience in my limited city practice with pet monkeys and performing monkeys, and I wonder what yours is.

DR. CAMPBELL: We use grapes a great deal, and things like that. You have to use all tricks and wiles to get ahead of monkeys. Don't think you can catch a monkey with two, four or five grains. You have got to give him ten or fifteen to get him so that you can handle him.

Now, about a baboon on which I did an enterotomy. We put him in the shifting-cage and administered two ounces of chloroform, which was sufficient. In a week, when we decided to take off the bandages, we had to use a pound and a half to get him under.

MEMBER: We have a number of monkeys with intestinal disorders and we find that these monkeys should be quarantined in the hospital.

DR. CAMPBELL: Yes, if it is necessary to put them in the hospital. We don't know how to treat them for intestinal trouble. Some food derangement of course is causing it. What specimen do you refer to, doctor?

MEMBER: Small ringtail of South America.

DR. H. V. CARDONA: You mentioned bandaging a monkey. How do you keep the bandage in place?

DR. CAMPBELL: The animal is completely under anesthesia. The bandage is brought right up around and in between the hind legs and then wrapped around the body and sewn tight. If he interferes with the bandages, we grab that monkey again and put him under morphin. However, in several cases of baboons that I have done, they haven't touched the bandage at all and the wound was nice and dry.

DR. CARDONA: I have had trouble in maintaining the bandage there and really I have hardly been able to find a way of forcing them to keep the bandage in place except by giving them hypnotics. For preventing mutilation after any surgical procedure, what system do you use?

DR. CAMPBELL: When working with hyenas, wolves, or bears, that bite the ears because of flies, we put them in collars made of tin, and also part of them with galvanized iron collars—big iron collars that stick out and prevent their reaching the infected parts.

DR. CARDONA: In connection with the feeding in zoölogical gardens, there seems to be a tendency on the part of the attendants, primarily the Board of Park Commissioners, to think that the veterinarian knows very little in connection with wild life. Do you have any idea where a veterinarian who has become interested in wild life could find textbooks by some good authority? Of course, I know that we have those authorities mostly in England. Dr. Fox, of Philadelphia, doesn't say very much on the feeding of most of the specimens we have.

DR. CAMPBELL: The discovery of the food factors, vitamins and minerals, has brought about a big step in the feeding of wild animals. This has been one of the bugbears in so many specimens we have. They just lack the something that is required to keep them going and you know they are very prone to rickets, etc.

One of the big things in connection with the zoo is the feeding of vitamins, largely in the form of cod-liver oil, cabbage, carrots, tomato; all of these should be in the veterinary hospital menu. Give them something in the way of meat, or a dog to gobble up.

DR. CARDONA: You said that the best thing to do with the Primates when they have any disturbance of the respiratory tract is to leave them alone.

DR. CAMPBELL: Medication is worse than useless. Don't fight them. They may allow the medicine to trickle out of the sides of their mouths and you may have accomplished nothing.

DR. CARDONA: I might be wrong in my contention, but don't you think that for the self-preservation of the veterinarian, and to inspire a little more consideration for his ability on the part of the zoo-keeper, it is better to give the animal something.

DR. CAMPBELL: There is no getting away from that fact. After you get your zoo attendants to have confidence in you and know what you are going to do is right, they will stand by you I think. That is a very good point, however.

DR. CARDONA: We have quite a number of parrots out in my country and their principal diet is sunflower seeds. Could you give me an idea if that is a practical ration for parrots, or do you advise the use of insects, etc.?

DR. CAMPBELL: Apparently we go back to experience with parrots. Sunflower seed seems to be the one thing they do well on. Supplement it with a little carnivorous food and a little fruit occasionally—bananas, tomatoes, and things like that, but not too much in the way of carnivorous food.

My experience, along with the reading of many books on the subject of diseases of birds, has led me to believe that the feeding of too much meat creates a craving for them to mutilate themselves.

DR. J. H. GILLMANN: I would like to know the average life of the common zoölogical garden animal.

DR. CAMPBELL: Contrary to the general opinion, the elephant, which is supposed to live a thousand years or something like that, lives just an ordinary span of life. We have no record of an elephant living longer than 50 years. That is, away from its home, it does not. Even in India, the home of the elephant, the record is not authentic that they live to be 90 to 100 years of age. In this country there is no record of an elephant living over 50 years.

No animal lives longer than man. The lion lives 20 or 25 years. The span of life varies quite a bit in small animals, which have a shorter duration of life. Horses live to 30 or 35 years. Zebra and deer live about that long. We have had sacred cattle live 25 years. However, the life of the zoölogical garden animal is going to be better from now on, with a better system of eating and better understanding of feeding vitamins and minerals. Birds will live quite a long time. Swans live considerably longer than smaller birds.

MEMBER: We have considerable trouble with monkeys losing hair.

DR. CAMPBELL: That is common in monkeys that have just come over and are not acclimated.

MR. L. J. BROSEMER: I have heard one of the doctors ask if there is any textbook obtainable on wild animals. About two years ago, we turned to the English, French and German literature for all practical works done with animals and to date we have compiled about 1,300 pages. As you probably know, we charge nothing for this and we would be glad to share it with anyone who cares for it. Quite a bit of time and the expenditure of thousands of dollars went into this research and so far no one has made any use of it. Anyone is welcome to it.

DR. H. H. BAUR: The monkeys at our local zoo will fight between the cages and bite the fingers of each other. Before I can stop that, infection starts and I have to amputate. Have you anything to recommend?

DR. CAMPBELL: That is what you have to do in the case of a finger or tail. They stick a finger or the tail into the adjoining cage and the first thing you know it is either bitten off or mutilated so that it must be amputated. What we do is to put a protective screen around so that they can't start trouble in the next cage. When they do, however, the only thing is amputation.

One thing about the monkey is that his mentality is such that in case of infection or irritation he will persist in chewing on the part and you have got to collar him, and in many cases it is advisable to get rid of him.

DR. QUITMAN: Pardon me, Dr. Campbell, if I make a suggestion or reply to Dr. Cardona's inquiry regarding the maintenance of bandages on these animals.

. . . Here Dr. Quitman described by illustration his method of bandaging small animals. . . .

One other suggestion: You all know how hard it is to maintain a bandage around a cat where you use a roller bandage, and you know many dogs will attempt to interfere with the bandage as soon as they come out from the anesthetic.

One way of preventing the dog from interfering with the bandage, or the bandage slipping off the cat is by this method: I keep constantly on hand the twelve-inch wide by five-yard long adhesive plaster and tear off a strip wide enough to cover fully the wound and dressing, and wind it around that animal. It sticks on the coat and I let it overlap upon itself so that if the coat is wet, as it is likely to be immediately after the operation, it will stick in place.

Something peculiar about dogs is that ordinarily if they will permit a bandage to be maintained on them any length of time, they will

never interfere with that bandage. I don't believe I have more than one dog in a hundred that will ever interfere with the bandage or dressing that is maintained by the adhesive plaster entirely around the body or limb.

In bandaging the dog's foot—you know how he fusses with that—make your regular dressing and then over that the strip of adhesive plaster is wound so that the strips will cross each other, and be careful not to cover the paws, then he will not interfere with it.

In dressing the paws, and that would apply to any wild animal, be careful not to compress or interfere with the claws. If you bind them down, the animal is more liable to fuss with that bandage.

I should think that the adhesive plaster dressing to maintain the dressing would work nicely in zoölogical garden animals, too, doctor. I know that in the case of a monkey, he let it alone nicely. He thinks it a decoration of some kind.

Now, speaking of setting fractured legs of birds, small birds. In city practice, of course, we get mostly birds like the canary. In broken limbs, it is surprising, as Dr. Campbell stated, how nicely they do. I have had cases of broken bones in birds such as the canary, where they just hang by the tiniest shred of skin. One could not conceive that they could possibly heal, yet they do, perfectly.

My method of setting the broken bones in the legs of small birds is this: I take an ordinary quill toothpick, cut the sharp point off, immerse it in hot water until it becomes flexible, spring it around the leg; after, of course, reducing the fracture, and the quill will curve right up on that leg. Then you can maintain top and bottom by a little narrow strip of plaster. If it happens to be a large bird you can use two or three of these quills. Let them overlap on themselves. You will find that they make the nicest kind of a cast for a small bird.

Taking No Chances

Much and loud protest against all official precautions to prevent spread of the rabies in Seattle has been voiced by Mr. Trent, president of a local anti-vivisection society. Mr. Trent has insisted that there's no such thing as rabies here or anywhere and, by inference, has upheld the right of dogs to bite whomsoever they please. Dr. Frank Carroll, city health commissioner, invites Mr. Trent to come around when the next rabies-suspect dog is picked up and be bitten in person, but Mr. Trent indignantly declines. Opportunity for fair test is thus evaded; but Mr. Trent's judgment is to be commended. It's easy to talk, but it would be hard luck for an anti-vivisectionist to be bitten by a dog that might happen to be really mad.

Seattle (Wash.) Daily Times.

A Long Jump

"Did you educate that flea yourself?" asked the amazed onlooker of the owner of the performing insect.

"Yes, I raised him from a pup."

CANINE RABIES EXPERIMENTAL VACCINATION*

Second and Third Reports

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This third report is designated "Second and Third Reports" because it involves and supplements the second report which was presented at the Veterinary Conference in Philadelphia, in January, 1932, but which was not published.

Our first report[1] contained a description of the purpose of our experiments, the location and description of the grounds and buildings, the sources of the dogs used and susceptibility tests, and a preliminary report of the results of the use of carbolized canine rabies single-dose vaccines obtained from four different commercial laboratories. These four vaccines, in order not to disclose the identities of the laboratories from which they were obtained, were designated as vaccines A, B, C and D.

The first report contained the following summary:

Of 27 dogs vaccinated and later exposed, 24 (89 per cent) died of rabies.

TABLE I—*Summary of results of the use of carbolized vaccine.*

TREATMENT	DOGS	DIED			ALIVE
		RABIES	RABIES NOT PROVED	OTHER CAUSES	
Vaccine A	10	7	2	1	0
Vaccine B	10	9	0	0	1
Vaccine C	10	8	0	1	1
Vaccine D	10	9	0	0	1
Totals	40	33 (82.5%)	2	2	3
Controls	16	13 (81.25%)	1	1	1

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

Of 11 unvaccinated controls exposed, 10 (91 per cent) died of rabies.

Since the report was made, the remainder of the 40 vaccinated dogs and additional controls were exposed, and the final summary of the results of dogs treated with carbolized vaccine is presented in table I.

Discussion

The results of our studies, in the use of the single-dose carbolized canine rabies vaccine, indicated that none of the vaccines used were capable of immunizing dogs against any of the viruses used.

There was some indication that the injection of street virus did not immunize dogs against a subsequent injection, 60 or more days later, of either the same strain or a different strain of virus.

The single-injection method of vaccinating dogs with carbolized vaccine is apparently unreliable and does not immunize dogs against rabies, and, therefore, should not be relied upon as a means of controlling rabies in dogs.

Chloroform-Treated Vaccine

After the first experiments in the use of carbolized vaccines were nearing completion, another group of healthy dogs was inoculated by different channels—subarachnoid, intramuscular and intravenous—with various dilutions and various-sized doses of rabies virus to determine, if possible, the comparative minimum fatal dose and to form a better basis for exposing dogs after vaccination with a chloroform-treated vaccine. In these tests, only one virus (27039) was used and dilutions were made so that 0.5 cc represented the dose in each case. Emulsions ranging from 10.0 per cent down to 0.009 per cent were used, and the 0.5-cc dose contained amounts of brain material ranging from 0.8 grains (50 mg) down to approximately 0.0,007 grains (0.05 mg). The results were somewhat irregular but there seemed to be some indication, within certain limits, that the dispersion of virus by dilution, rather than the amount of virus, had some relation to infections which took place.

On August 20, 1931, 20 healthy dogs were vaccinated with commercial (Laboratory A) chloroform-treated rabies vaccine prepared by the Kelser method. An equal number of dogs were set aside as controls. These groups were subdivided and, later, individual dogs or groups were exposed through different channels to different strains of virus as is shown in the accompanying tables.

TABLE II—*Street virus 50012D454 (10 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (CC)	
242	8–20–31	11–16–31	Subarachnoid	0.1	Lived
452			Subarachnoid	0.2	Lived
195			Intramuscular	0.2	Lived
318			Intramuscular	0.5	Lived
453			Intramuscular	1.0	Lived
223	Controls		Subarachnoid	0.1	R.* 22 days
214			Subarachnoid	0.2	R.* 21 days
297			Intramuscular	0.2	D. † 10 days
258			Intramuscular	0.5	Lived
436			Intramuscular	1.0	Lived

*Rabies.
†Died. Rabies not proved.

TABLE III—*Street virus 51371 (10 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (CC)	
178	8–20–31	11–16–31	Subarachnoid	0.1	R.* 15 days
300			Subarachnoid	0.2	R.* 19 days
237			Intravenous	0.5	Lived
299	Controls		Subarachnoid	0.1	R.* 18 days
366			Subarachnoid	0.2	R.* 16 days
277			Intravenous	0.5	Lived

*Rabies.

TABLE IV—*Pasteur virus 1850EE5 (10 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (CC)	
248	8–20–31	11–17–31	Subarachnoid	0.1	R.* 9 days
228			Subarachnoid	0.2	R.* 9 days
174			Intravenous	0.5	Lived
290	Controls		Subarachnoid	0.1	R.* 8 days
353			Subarachnoid	0.2	R.* 8 days
391			Intravenous	0.5	Lived

*Rabies.

TABLE V—*Street virus 27039B71 (10 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (cc)	
184	8–20–31	11–17–31	Subarachnoid	0.1	R.* 12 days
308			Subarachnoid	0.2	R.* 15 days
381			Intravenous	0.5	Lived
420			Intramuscular	1.0	Lived
350	Controls		Subarachnoid	0.1	R.* 16 days
351			Subarachnoid	0.2	R.* 14 days
331			Intravenous	0.5	Lived
307			Intramuscular	1.0	R.* 52 days

*Rabies.

TABLE VI—*Street virus 50012D223 and 214 (10 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (cc)	
185	8–20–31	12–17–31	Subarachnoid	0.1	R.* 19 days
362			Subarachnoid	0.2	Lived
285	Controls		Subarachnoid	0.1	R.* 21 days
284			Subarachnoid	0.2	R.* 16 days

*Rabies.

TABLE VII—*Street virus 27039B82 (1 per cent emulsion).*

DOG	VACCINATED	VIRUS EXPOSURE			RESULT
		DATE	METHOD	DOSE (cc)	
292	8–20–31	4–15–32	Intramuscular	2.0	Lived
364			Intramuscular	4.0	Lived
192			Intramuscular	4.0	Lived
361	Controls		Intramuscular	2.0	Lived
377			Intramuscular	4.0	Lived
294			Intramuscular	4.0	Lived

TABLE VIII—*Summary of results of initial exposures (tables II to VII inclusive).*

		VACCINATED	CONTROLS
Dogs		20	20
Died of rabies	No.	7	11
	%	35	55
Died (rabies not proved)	No.	0	1
	%	0	5
Lived	No.	13	8
	%	65	40

INCUBATION PERIODS FOLLOWING ARTIFICIAL EXPOSURES

The average period of time from exposure until death in 117 proved cases of rabies in dogs was 21.5 days. The usual period was between 13 and 25 days.

In 13 dogs (table XII) showing a period of 30 days or more, the average was 59.8 days.

One dog showed a period of 223 days.

Twelve dogs showed an average of 46.3 days.

In 104 dogs showing a period of less than 30 days, the average was 16.7 days. Four of these were exposed to fixed virus and died in an average of 8.5 days.

In 100 dogs showing a period of less than 30 days, and which were exposed to street virus, the average period until death was 17 days.

DISCUSSION

There undoubtedly has been a greater increase in the number of centers of infection throughout the country, and the number of cases of rabies both in animals and in humans, since the single-dose rabies vaccine came into use, than for any other equal period of time known; yet the states are better equipped with transmissible disease control organizations for combating animal diseases. This increase may be partly due to a feeling of false security built up through the use of the single-injection vaccine.

We are convinced that the use of the canine rabies vaccines now on the market does not offer a successful means for the control and suppression of rabies.

If sanitary officials are going to cope successfully with the rabies situation to prevent an increase in the number of centers

TABLE IX—*Results of subsequent exposures of survivors of previous experiments.*

Vaccinated 8-20-31							Controls						
Dog	Date	Virus*	Emulsion (%)	Dose (cc)	Method†	Result	Dog	Date	Virus	Emulsion (%)	Dose (cc)	Method	Result
242	11-16-31	50012	10	0.1	SA		223	11-16-31	50012	10	0.1	SA	R.‡ 22 days
	7-21-32	57382	1	0.2	SA								
	10-14-32	58982	10	0.2	SA								
	6-26-33	58982	10	0.25	SA	Alive 8-7-33							
452	11-16-31	50012	10	0.2	SA		214	11-16-31	50012	10	0.2	SA	R.‡ 21 days
	7-21-32	57384	1	0.2	SA								
	10-14-32	58982	10	0.2	SA								
	6-26-33	58982	10	0.25	SA	Alive 8-7-33							
195	11-16-31	50012	10	0.2	IM		297	11-16-31	50012	10	0.2	IM	D.(?)§ 10 days
	4-15-32	B82	1	2.0	IM								
	7-20-32	B88	1	0.2	SA								
	10-14-32	58982	10	0.2	SA	D.(?)§ 11-15-32							
318	11-16-32	50012	10	0.5	IM		258	11-16-31	50012	10	0.5	IM	
	4-15-32	B82	1	2.0	IM			4-15-32	B82	1	2.0	IM	
	7-20-32	B88	1	0.2	SA			7-20-32	B88	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			10-14-32	58982	10	0.2	SA	
	6-26-33	58982	10	0.25	SA	Alive 8-7-33		6-26-33	58982	10	0.25	SA	Alive 8-7-33
453	11-16-31	50012	10	1.0	IM		436	11-16-31	50012	10	1.0	IM	
	4-15-32	B82	1	2.0	IM			4-15-32	B82	1	2.0	IM	
	7-20-32	B88	1	0.2	SA			7-20-32	B88	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			10-14-32	58982	10	0.2	SA	
	6-26-33	58982	10	0.25	SA	Alive 8-7-33		6-26-33	58982	10	0.25	SA	Alive 8-7-33

Note: Previous history of dogs 242, 452, 195, 318, 453, 223, 214, 297, 258 and 436 given in table II.

TABLE IX—*Results of subsequent exposures of survivors of previous experiments—Continued.*

Vaccinated 8–20–31							Controls						
Dog	Date	Virus*	Emulsion (%)	Dose (cc)	Method†	Result	Dog	Date	Virus	Emulsion (%)	Dose (cc)	Method	Result
237	11-16-31	51371	10	0.5	IV		277	11-16-31	51371	10	0.5	IV	
	4-15-32	51371R	2.5	1.0	IV			4-15-32	51371R	2.5	1.0	IV	
	7-21-32	57384	1	0.2	SA			7-21-32	57384	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			10-14-32	58982	10	0.2	SA	D.(?)§ 12-14-32
	6-26-33	58982	10	0.25	SA	Alive 8-7-33							
174	11-17-31	1850EE5 (Pasteur)	10	0.5	IV		391	11-17-31	1850EE5 (Pasteur)	10	0.5	IV	
	4-15-32	1850EE5 (Pasteur)	2.5	1.0	IV			4-15-32	1850EE5 (Pasteur)	2.5	1.0	IV	
	7-21-32	57384	1	0.2	SA			7-21-32	57384	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			10-14-32	58982	10	0.2	SA	
	6-26-33	67074	10	0.25	SA	Alive 8-7-33		6-26-33	67074	10	0.25	SA	Alive 8-7-33
381	11-17-31	B71	10	0.5	IV		331	11-17-31	B71	10	0.5	IV	
	4-15-32	B82	1	1.0	IV			4-15-32	B82	1	1.0	IV	
	7-20-32	B88	1	0.2	SA			7-20-32	B88	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			10-14-32	58982	10	0.2	SA	
	6-26-33	67074	10	0.25	SA	R.‡ 13 days		6-26-33	67074	10	0.25	SA	Alive 8-7-33
420	11-17-31	B71	10	1.0	IM		307	11-17-31	B71	10	1.0	IM	R.‡ 52 days
	4-15-32	B82	1	2.0	IM								
	7-20-32	B88	1	0.2	SA								
	10-14-32	58982	10	0.2	SA	R.‡ 16 days							

Note: Previous history of dogs 237 and 277 given in table III; of dogs 174 and 391 in table IV; of dogs 381, 420, 331 and 307 in table V.

TABLE IX—*Results of subsequent exposures of survivors of previous experiments—Concluded.*

Vaccinated 8-20-31							Controls						
Dog	Date	Virus*	Emulsion (%)	Dose (cc)	Method†	Result	Dog	Date	Virus	Emulsion (%)	Dose (cc)	Method	Result
362	12-17-31	50012	10	0.2	SA		284	12-17-31	50012	10	0.2	SA	R.‡ 16 days
	7-21-32	57384	1	0.2	SA								
	10-14-32	58982	10	0.2	SA								
	6-26-33	67074	10	0.5	SA	Alive 8-7-33							
292	4-15-32	B82	1	2.0	IM		361	4-15-32	B82	1	2.0	IM	
	7-20-32	B88	1	0.2	SA			7-20-32	B88	1	0.2	SA	
	10-14-32	58982	10	0.2	SA			11- 2-32	58082	10	0.2	SA	D.(?)§ 11-24-32
	6-26-33	67074	10	0.25	SA	Alive 8-7-33							
364	4-15-32	B82	1	4.0	IM		377	4-13-32	B82	1	4.0	IM	
	7-20-32	B88	1	0.2	SA			7-20-32	B88	1	0.2	SA	
	10-14-32	58982	10	0.2	SA	R.‡ 17 days		11- 2-32	58982	10	0.2	SA	D.(?)§ 11-16-32
192	4-15-32	B82	1	4.0	IM		294	4-15-32	B82	1	4.0	IM	D. during inoculation
	7-20-32	B88	1	0.2	SA			7-20-32					
	10-14-32	58982	10	0.2	SA								
	6-26-33	67074	10	0.25	SA	Alive 8-7-33							

Note: Previous history of dogs 362 and 284 given in table VI; of dogs 292, 364, 192, 361, 377 and 294 in table VII.
*B71, B82, etc. = 71st or 82nd passage through rabbits.
†SA = Subarachnoid; IM = Intramuscular; IV = Intravenous.
‡Rabies.
§(?) = Rabies not proved.

TABLE X—*(Table IX condensed). Results of subsequent exposures of survivors of previous experiments.*

VACCINATED 8–20–31					CONTROLS				
DOG	TABLE	EXPOSURES		RESULT	DOG	TABLE	EXPOSURES		RESULT
		NUMBER	LAST				NUMBER	LAST	
242	II	4	6-26-33	Alive 8-7-33	223	II	1	11-16-31	R.* 22 days
452	II	4	6-26-33	Alive 8-7-33	214	II	1	11-16-31	R.* 21 days
195	II	4	10-14-32	D.(?)† 11-15-32	297	II	1	11-16-31	D.(?)† 11-26-31
318	II	5	6-26-33	Alive 8-7-33	258	II	5	6-26-33	Alive 8-7-33
453	II	5	6-26-33	Alive 8-7-33	436	II	5	6-26-33	Alive 8-7-33
237	III	5	6-26-33	Alive 8-7-33	277	III	4	10-14-32	D.(?)† 12-14-32
174	IV	5	6-26-33	Alive 8-7-33	391	IV	5	6-26-33	Alive 8-7-33
381	V	5	6-26-33	R.* 13 days	331	V	5	6-26-33	Alive 8-7-33
420	V	4	10-14-32	R.* 16 days	307	V	1	11-17-31	R.* 52 days
362	VI	4	6-26-33	Alive 8-7-33	284	VI	1	12-17-31	R.* 16 days
292	VII	4	6-26-33	Alive 8-7-33	361	VII	3	11- 2-32	D.(?)† 11-24-32
364	VII	3	10-14-32	R.* 17 days	377	VII	3	11- 2-32	D.(?)† 11-16-32
192	VII	4	6-26-33	Alive 8-7-33	294	VII	2	7-20-32	D. during inoculation

*Rabies.
†(?) = Rabies not proved.

TABLE XI—*Summary*

	VACCINATED		CONTROLS	
	No.	%	No.	%
Dogs	20		20	
Died of other causes (definite)	0		1	
Balance	20		19	
Died—rabies proved	10	50	11	58
Rabies symptoms; Negri bodies not found; inoculated rabbits survived more than 100 days; rabies not proved	1		4	
Total died	11	55	15	79
Alive 8-7-33	9	45	4	21

of infection, they must apply more strict sanitary police measures.

Our latest experiments have indicated a possibility of the chloroform-treated vaccine being somewhat effective as an immunizing agent against certain strains of street virus. There possibly has been some indication that exposure to some strains of street virus builds some resistance to later exposures. Generally speaking, however, dogs which have resisted exposure to a comparatively large dose of a potent virus have later succumbed to rabies infection when exposed to a comparatively small dose of virus. Some dogs succumbed after resisting several exposures.

TABLE XII—*Data on 13 dogs with incubation period of 30 days or more.*

DOG	EXPOSED	VIRUS	METHOD	DOSE (CC)	DIED	DAYS
18	11-20-28	27993	Intramuscular	1.0	12-21-28	31
25	11-20-28	27993	Intravenous	0.25	12-20-28	30
33	11-20-28	27993	Intraperitoneal	0.25	1- 6-29	47
21	11-20-28	27993	Intravenous	1.0	12-30-28	40
95	11-20-28	27993	Intravenous	0.5	12-29-28	39
108	11-20-28	27993	Intramuscular	0.25	12-30-28	40
81	4-18-29	29995	Dog bite	....	6-30-29	73
239	4-19-29	29952	Subarachnoid	0.25	6-13-29	55
240	4-19-29	27039*	Subarachnoid	1.0	7- 8-29	80
307	11-17-31	B71	Intramuscular	1.0	1- 8-32	52
357	7-20-32	B88	Subarachnoid	0.2	8-24-32	35
232	9-22-32	58982	Subarachnoid	0.2	5- 3-33	223
262	11- 2-32	58982	Subarachnoid	0.2	12- 5-32	33

*Submaxillary gland extract.

TABLE XIII—*Data on rabbits with prolonged incubation periods.*

RABBIT	EXPOSED	VIRUS	METHOD	DOSE (CC)	DIED	DAYS
7190	11–20–28	27993	Intraocular	0.25	2–14–29	86
7198	11–22–28	27993	Subdural	0.25	12–29–28	37
7288	12–28–28	27993	Subdural	0.25	7–22–29	206
7290	12–28–28	27993	Subdural	0.25	2– 3–29	37
7594	3–15–29	29870	Subdural	0.25	5–31–29	77
8294	11–20–29	33795	Subdural	0.25	12–29–29	39
10499	11–17–31	50012	Subdural	0.20	1–22–32	66
10503	11–17–32	50012	Subdural	0.20	2–16–32	91
10996	4–15–32	1850EE	Subdural	0.25	6–13–32	59
12193	5– 5–33	58982D232	Subdural	0.25	7–24–33	80

A few dogs in both the vaccinated and control groups have resisted numerous exposures. It seems that if a dead virus will successfully immunize against rabies, a stronger resistance should exist in dogs which have survived successive exposures to a living virus.

Quite frequently cases of rabies have been reported in vaccinated dogs which were exposed to the bite of a rabid dog previous to vaccination, but very little data have been furnished on dogs which have been exposed to natural infection subsequent to vaccination.

In our experiments, all inoculated dogs were kept in individual locked cages made of wire cloth. No cases of rabies developed in healthy dogs which were placed in cages previously occupied by rabid dogs, and no cases developed in healthy dogs kept in separate cages in close contact with cages containing dogs showing physical symptoms of rabies.

There was no evidence in any case of harmful effects following the use of vaccine. All vaccinated dogs remained healthy from the standpoint of rabies until after exposure to rabies virus.

CONCLUSIONS

Conclusions at this time can be no more than tentative.

Our experiments indicated that the carbolized vaccines used did not immunize against rabies.

The experimental use of chloroform-treated vaccine has offered somewhat more encouraging results than the carbolized vaccine, but not sufficient to warrant confidence in it to the exclusion of sanitary and police measures. Apparently no harmful effects are caused by the vaccine, and it is doubtful if enough good can come from its use to justify the expense involved.

Sanitary officials should not rely on vaccination as a means of rabies control.

Effective quarantine has successfully controlled rabies in districts where enforced.

All dogs known to have been exposed to rabies should be killed.

The usual 100-day quarantine does not furnish positive assurance that cases of rabies will not develop later, although for practical purposes it probably should not be increased.

There undoubtedly is a successful way of immunizing dogs against rabies, but neither the proper method nor the proper vaccine seems to have been found.

Several directions of future study have been indicated by past results, and we hope to continue our work on canine diseases, but we are not encouraged with the results obtained in testing the immunizng properties of canine rabies vaccines now on the market.

REFERENCE

[1]Barnes, M. F., Metcalfe, A. N., and Lentz, W. J.: Investigations of canine diseases with special reference to rabies. Preliminary report. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (1), pp. 34-52.

(Note: For discussion of this paper, see page 756. The two papers on rabies vaccine were discussed jointly.)

U. S. Specialists Proficient in Chick Sexing

So-called chick sexing—picking out the pullets and cockerels in a group of newly hatched chicks—has recently been done with considerable accuracy by poultry specialists of the U. S. Department of Agriculture. The method, first developed by the Japanese, consists of observation of the size and shape of the genital eminence, variations between the sexes being only slight at this early age. After a study covering four months, the Department specialists acquired an accuracy of more than 90 per cent, checking the results by postmortem examination of the chicks. Accuracy of 70 per cent was acquired quickly by those doing the work, but much practice was necessary to pass the 90 per cent point.

A mimeographed pamphlet which gives the essential directions has been published by the Department. Copies may be obtained from the Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

12th International Veterinary Congress
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RABIES VACCINE PROTECTION TEST*

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Many attempts have been made to establish the potency and efficacy of rabies vaccine by animal protection tests, which generally fail to yield consistent results, because of difficulties experienced with the infective dose. The dose usually proves too severe or innocuous, depending on the amount used and the mode of injection. The intracranial injection invariably proves fatal, regardless of the variation in the size of the dose which will infect, and the same applies to the intraocular injection. Other modes of infection, the subcutaneous, intravenous or intramuscular, and natural or artificial exposure, are so uncertain that similar results cannot be obtained repeatedly with any degree of consistency.

The intracranial injection is most direct, and the intraocular only a trifle less so, from the viewpoint of the distance the virus has to travel from the injection point to the brain tissue. A little further removed, however, is the oral region and it is for this reason that we selected the tongue as the repository for the infective dose, which resulted in the establishment of the intralingual method of injection.

The method consists in the proper restraint of the animal, best accomplished by anesthesia or narcosis. The rabbit may be placed in a box of suitable size, with a hole at one end allowing for the protrusion of the head and then, by the use of a mouth speculum or rings with chains, the upper and lower jaws can be held apart and the tongue grasped with a pair of forceps for the injection. We prefer to inject 0.1 cc of a 5 per cent emulsion of brain-tissue virus moderately deep into the tongue muscles, off to one side of the median line, so that a bleb is noticeable beneath the surface. The animal apparently is not inconvenienced immediately or later by the deposition of the virus and the period of incubation following the method of injection is only slightly longer than that following the intracranial injection. In two sets of rabbits, the one injected intracranially averaged a period of incubation of 6 to 7 days, while the other set, injected with the same dose of fixed rabies virus, averaged 8 to 9 days. Of the total number injected intralingually, 20 per cent survived, while of an equal

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

number injected intracranially, 100 per cent promptly developed rabies. Only occasionally will an older rabbit survive the intracranial infective dose.

The rabies vaccine protection test is conducted by the injection of three, five or more rabbits with the test vaccine subcutaneously, followed 21 days later with an injection of the infective dose intralingually, whereupon the test rabbits and an equal number of virus-injected control rabbits are held under observation for 14 days.

The preparation of the infective dose of fixed rabies virus is an important detail of the protection test. The rabbit-brain-tissue "seed" virus, usually kept in 50 per cent glycerin, is emulsified and injected intracranially into a rabbit and, when the rabbit is moribund with rabies, so timed that this will occur on the day the infective dose falls due, the rabbit is chloroformed, the brain removed, immediately ground and made up in a 5 per cent brain-tissue emulsion in normal salt solution. The emulsion is strained through a 60-mesh silk and promptly used in 0.1-cc infective doses injected intralingually.

Table I shows the results of the rabies vaccine protection test applied on a batch of rabies vaccine consisting of brain and spinal cords of nine rabbits moribund with rabies, following the intracranial injection of fixed rabies virus. The fresh tissue virus emulsion was made up in a 50 per cent emulsion in normal salt solution and divided into five equal parts.

TABLE I—*Rabies vaccine protection test.*

GROUP	RABBITS	INJECTED WITH		INTERVAL	INFECTIVE DOSE	RESULTS
		MATERIAL	DOSE			
1	3	Rabies vaccine (A) (live*)	5 cc subcutaneously	22 days	0.1 cc of 5 per cent brain-tissue rabies virus (rabbit) intralingually	Living 100%
2	3	Rabies vaccine (B) (Chloroform-killed*)				Living 33⅓% Dead† 66⅔%
3	3	Rabies vaccine (C) (Phenol-killed*)				Living 100%
4	3	Rabies vaccine (D) (Formalin-killed*)				Living 33⅓% Dead† 66⅔%
5	3	Rabies vaccine (E) (Autoclave-killed*)				Dead† 100%
6	6	Controls				Living 33⅓% Dead† 66⅔%

*Rabies vaccine 33⅓ per cent brain tissue (rabbit).
†Deaths preceded by definite symptoms of rabies.

(A) Live vaccine: 40 cc of 50 per cent emulsion to which 20 cc of normal salt solution was added. Placed in cold storage for 44 days.

(B) Phenol-killed: 40 cc of 50 per cent emulsion to which 0.3 per cent of 90 per cent phenol was added. Held at room temperature for 44 days, when 10 cc of salt solution was added.

(C) Chloroform-killed: 40 cc of 50 per cent emulsion to which were added 20 cc of salt solution and 0.6 cc of chloroform. The material was held at room temperature for 44 days.

(D) Formalin-killed: 40 cc of 50 per cent emulsion to which 0.2 cc of formalin was added. Held at room temperature 44 days, when 10 cc of normal salt solution was added.

(E) Autoclaved virus: 40 cc of 33⅓ per cent emulsion was autoclaved at 15 pounds for one-half hour and held at room temperature for 44 days.

That Rabies Vaccine (A) (live) consisted of live rabies virus is borne out by the prompt development of rabies in two rabbits injected intracranially. Rabies Vaccines (B), (C) and (D) also were tested for infectivity by the intracranial injection of three rabbits with each lot. All of the rabbits continued apparently normal. Vaccine (E) was not tested for infectivity.

The three rabbits in group 1 (table I) survived the immunizing dose of (A), injected subcutaneously, and 22 days later survived the infective dose injected intralingually. Group 3 rabbits, injected with Rabies Vaccine (C) (phenol-killed) showed the same protection while groups 2 and 4 failed to the same extent, showing end results no better than the control group. Group 5 failed completely.

The tests indicate protection with the phenol-killed portion (C) equal to "live vaccine" (A), with results in the other groups no better than in the controls.

TABLE II—*Rabies vaccine protection test.*

RABBITS	INJECTED WITH		INTERVAL	INFECTIVE DOSE	RESULTS
	MATERIAL	DOSE			
5	Rabies vaccine 67819 (Chloroform-killed*)	5 cc subcutaneously	19 days	0.1 cc of 5 per cent brain-tissue rabies virus (rabbit), intralingually	Living 100%
7	Controls				Living 14% Dead† 86%

*33⅓ per cent brain-tissue rabies vaccine (horse).
†Deaths preceded by definite symptoms of rabies.

Table II shows the results of a single test on chloroform-killed rabies vaccine 67819—33⅓ per cent brain-tissue rabies vaccine (horse), with 100 per cent survivals, while 86 per cent of the controls died.

Tables III and IV show the results of rabies vaccine protection tests of three lots of rabies vaccine on rabbits (table III) and dogs (table IV) for the purpose of comparing the tests on rabbits with the test on dogs. The results are practically the same for chloroform-killed rabies vaccines 71902 and 72228, on rabbits and dogs. In both tests, 60 per cent or more of the vaccinated

TABLE III—*Rabies vaccine protection test.*

GROUP	RABBITS	INJECTED WITH		INTERVAL	INFECTIVE DOSE	RESULTS
		MATERIAL	DOSE			
1	3	Rabies vaccine 71902 (Chloroform-killed*)	5 cc subcutaneously	21 days	0.1 cc of 5 per cent brain-tissue rabies virus (rabbit), intralingually	Living 66⅔% Dead‡ 33⅓%
2	3	Rabies vaccine 72228 (Chloroform-killed†)				Living 66⅔% Dead‡ 33⅓%
3	3	Rabies vaccine 72289 (Formalin-killed†)				Living 33⅓% Dead‡ 66⅔%
4	5	Controls				Living 40% Dead‡ 60%

*33⅓ per cent brain-tissue rabies vaccine (bovine).
†33⅓ per cent brain-tissue rabies vaccine (horse).
‡Deaths preceded by definite symptoms of rabies.

TABLE IV—*Rabies vaccine protection test.*

GROUP	DOGS	INJECTED WITH		INTERVAL	INFECTIVE DOSE	RESULTS
		MATERIAL	DOSE			
1	5	Rabies vaccine 71902 (Chloroform-killed*)	5 cc subcutaneously	21 days	0.3 cc of 5 per cent brain-tissue rabies virus (rabbit), intralingually	Living 100%
2	5	Rabies vaccine 72228 (Chloroform-killed†)				Living 100%
3	5	Rabies vaccine 72289 (Formalin-killed†)				Living 40% Dead‡ 60%
4	5	Controls				Living 40% Dead ‡60%

*33⅓ per cent brain-tissue rabies vaccine (bovine).
†33⅓ per cent brain-tissue rabies vaccine (horse).
‡Deaths preceded by definite symptoms of rabies.

animals survived, while 60 per cent of the controls died. The formalin-killed rabies vaccine 72289 failed, as the number of deaths equaled those of the control animals.

SUMMARY

The intralingual injection of the infective dose of rabies virus makes it possible to obtain fairly consistent results in the potency testing of rabies vaccines. Rabbits are better suited for the test than dogs. The results of the rabbit and dog protection tests approximate each other closely. In both tests the end results are satisfactory when at least 60 per cent of the vaccinated animals survive, while 60 per cent or more of the controls died of the intralingual infective dose.

CONCLUSIONS

1. Formalin-killed and autoclave-killed rabies vaccines failed to pass the protection test, indicating that the formalin and heat similarly affected the potency of the products.
2. Chloroform-killed and phenol-killed rabies vaccines passed the protection test.
3. Live rabies vaccine passed the protection test.

DISCUSSION

DR. B. M. LYON: I particularly enjoyed these two papers on rabies. I would like to corroborate Dr. Reichel's statement on the variability of virus infectivity in conducting these tests. We did considerable work at our laboratory in 1921 and 1922, with the phenol-killed vaccine, and we arrived at the same conclusions, as did Dr. Reichel, that the variability of virus infectivity as well as individual susceptibility are factors of considerable importance in the interpretation of the test.

DR. C. E. COTTON: I do not know whether or not it is proper to interject this thought here, but I recall at the final session of the annual meeting of this Association in Los Angeles the Committee on Resolutions presented their report. This business session followed the reading of papers and did not adjourn until after midnight, at which time the Resolutions Committee presented the report and there were not more than 20 or 25 members present. They included in their report a resolution placing this Association on record as condemning the method of controlling rabies by muzzling and quarantine. The Chairman of the Committee stated that this resolution had been presented to them too late to be considered by the Committee. Had it not been for the objection of one or two members, this resolution would have been adopted by the small number of men as representing the opinion and the voice of the American Veterinary Medical Association.

I do not think we are in any position at present to adopt any method of vaccination in lieu of control measures. Control measures, while not 100 per cent effective in this country, are proving effective when properly enforced. Hawaii and the British Isles have been kept free of rabies many years as the result of their establishing proper quarantine for a period of time on imported dogs. Dr. Barnes made a statement that perhaps the 100-day period of quarantine is not sufficient, but he thought it might be for practical purposes. He cited one instance where

the incubation after the inoculation of virus was 223 days. The majority of states require a 90-day quarantine in territories where rabies appears. I think that with our present knowledge, the period of quarantine should be extended to four months at least..

DR. H. W. SCHOENING: I was quite interested in both of the papers on the subject of rabies vaccination, and I was particularly interested in some of the points Dr. Barnes brought out with reference to the fact that certain dogs, after exposure, apparently developed clinical evidence of the disease, but rabies could not be proved, either by microscopic examination or inoculation of rabbits.

I have noticed the same condition in some of the experimental work that I have done with rabies in dogs, and it is a rather peculiar thing. I personally believe that those animals actually do die of rabies, but, in those particular cases, we cannot demonstrate it. Just what the explanation is I do not know. But I have had this experience: On reinoculation, perhaps two or three additional inoculations, one rabbit may come down and show Negri bodies, but in some instances repeated inoculations fail to demonstrate the disease.

I was struck by the rather long incubation period in the rabbits coming down with rabies, that Dr. Barnes reported. As I recollect, they ran 50 and 90 days a good many times.

DR. BARNES: Those were exceptions. The average was about 17 days.

DR. SCHOENING: It has been our experience that the usual period is about 16 to 22 days.

DR. REICHEL: Along with these tests we have made others in connection with the multiple dose treatment. The results will be reported at the American Public Health Association in October. In that series of experiments, phenol-killed rabies vaccine is taken and a series of rabbits injected with vaccine over a period of seven days, another series over a period of 14 days, and another over a period of 21 days.

Multiple injections are better than a single injection. A 5-cc dose divided into five 1-cc doses results in more protection than the single 5-cc dose, and I am of the opinion that veterinarians are beginning to realize this in the field.

DR. W. G. HOLLINGWORTH: I would like to ask for information on one point in regard to the quarantining of dogs that have bitten people. The New York Sanitary Code says that all dogs that have bitten persons, whether in an infected territory or not, must be kept under observation for a certain length of time. At home we follow the instructions and quarantine the dog for three weeks. If there is a period, what is the limit?

DR. BARNES: It has been our experience that after dogs show symptoms of rabies, usually they will die within five or six days. I believe the bite is infective before symptoms are observable. A dog will not bite on account of rabies until he is developing the disease. I think ten days would be sufficient in a district where there never had been rabies.

DR. REICHEL: Isn't it true that when the dog has rabies at the time he bites an individual, the dog will not live more than ten days?

DR. BARNES: They usually do not live that long.

DR. M. L. MORRIS: Within a period of ten to twelve days, five definite cases of rabies were removed from a pack of approximately 250 fox hounds. A conference of veterinarians was called to determine the most feasible procedure. The point in question was: Should we or should we not use vaccine? What sanitary measures should be instituted to control the infection?

The State Health Department would destroy the entire group unless some definite action was taken. I opposed any plan which involved the life of this pack of hounds. All dogs were vaccinated. Sanitary meas-

ures consisted of the isolation of all quarrelsome males. These in turn were each given three to five consecutive doses of vaccine. The females were separated into groups of not more than two or three to a group. These dogs were given but a single injection. All dogs were vaccinated again in six months. The five hounds which were removed from the pack concluded the outbreak and we have had no recurrence of the disease.

DR. LOUIS LEONPACHER: We have found in our work that we have rabies outbreaks every two or three years, and it is up to the veterinarian to decide what measures to take. Our experience has been that dogs treated by the distemper method generally survive, but we have not tried this vaccination where you know hydrophobia is in a pack. Our luck with large animals has been very, very poor, and my personal explanation is that when a dog gets into an argument with a mule or horse or cow, he generally bites that animal. I think I have tried about eight times to give distemper injections to large animals and they always died as early as 12 to 18 days in spite of treatment, but the dogs treated stood up and did not show any trouble afterward. As far as quarantine measures, we always advise a maximum of five days. If a dog has bitten or scratched somebody, assuming the dog has hydrophobia, that dog will not live longer than 72 hours.

Boosting the Old Gray Mare

Here is some news that will be of interest to all who are anxious to see the horse restored to its rightful place on the farm. The Horse and Mule Association of America is conducting a nation-wide contest and offering prizes worth $2,000 for the best letters on "Why Horses and Mules Are the Best Farm Power."

The contest began the second week in April and will close on May 26. Those who are eligible to compete are farmers, farm boys and girls, merchants and bankers in farming communities, and county agents and vocational teachers of agriculture. No effort is being spared to make the contest a success. A series of 26 advertisements appearing in as many state and national farm papers, and broadcasts of the "Old Gray Mare" program from eight radio stations are arousing enthusiasm both for the contest and for the purpose back of it. Stations carrying the programs are: WLS, Chicago; WLW, Cincinnati; WOC-WHO, Des Moines; KTSP, Saint Paul; WFAA, Dallas; WSM, Nashville; KPRC, Houston; WKY, Oklahoma City.

Circulars that have gone out to agricultural communities emphasize this appeal: "Horse and mule power on farms means a better market for food crops . . . lower crop production costs for farmers . . . more 'able-to-buy' customers for merchants . . . an easier financing problem for banks. Join with us . . . boost farm-grown power that eats farm-grown feed . . . that wears harness made of leather from farm-grown steers, and sold by home-town merchants."

STUDIES ON THE COMMON COLD IN CHICKENS*

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Chickens are subject to an infectious condition of the nasal passages and sinuses that closely resembles the so-called common cold of man. Therefore, since the study of the human cold has been greatly impeded by the lack of a satisfactory experiment animal, it was thought that a study of this condition in chickens, in conjunction with investigations on the human cold, might prove of value.

When the investigation was undertaken, our object was to obtain a filtrable virus of the chicken cold that could be maintained in an active condition in tissue cultures. However, it was soon found impossible to obtain for the tissue cultures an infectious filtrate, so it was decided to concentrate our efforts upon determining, first, whether the causative agent of the chicken cold could be considered a filtrable virus and, second, whether it would be possible to isolate a cold-producing organism.

MATERIAL AND METHODS

In the preliminary experiments, small chickens were obtained from the market, isolated in sterile cages and kept under observation for several days. If no cold developed among the group, they were then used for inoculation experiments. However, the results obtained showed that such chickens are so prone to colds that they could not be used. Apparently all such chickens were about to develop a cold, had a cold, or had just recovered from a cold. Seventy-five per cent of them developed one following inoculation with non-infectious solutions, such as normal saline, broth or Tyrode's solution, while 50 per cent developed colds while kept under observation prior to inoculation. Many of the chickens that supposedly had recovered from these spontaneous colds suffered a relapse, while others exhibited a certain amount of resistance to inoculation of cold-producing material. Consequently, it became necessary to obtain healthy chickens that had not been exposed to colds.

For this reason the chickens used in the later experiments, reported here, were all obtained from poultry farms where "springers" were raised from incubated eggs. These chicks were

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kept elevated from the ground, free from droppings, and fed on a special food containing cod-liver oil. When the chicks were three to six weeks old, they were transferred directly to our laboratory. Here the special food was continued, in order to prevent confusing results with avitaminosis, a condition showing similar symptoms, found by J. R. Beach,[1] and later by Seigfried,[2] to be brought about in chickens by lack of proper diet. In order to keep the chickens dry and clean, they were housed in sterilized coops having a one-inch mesh wire bottom, with the food and water containers fastened on the outside. The cages were sterilized by painting them with a 1:1,000 solution of bichlorid of mercury, and then washing them with live steam. No vermin were present. The room was kept as nearly as possible at a temperature approximately 68° F. with the windows open day and night. After entering our laboratory the chickens were kept under observation for several days before being used for experimental purposes. Of the 500 chickens obtained from these farms, only 18 developed spontaneous colds.

A number of strains of cold-producing material was used in these experiments. The infectious material was readily obtained from market chickens suffering with colds. Each chicken carrying the cold was purchased at a different time and from a different dealer, all but one being successfully transmitted from one animal to another by inoculation. One of the transmitted strains was carried for two months, through 20 passages; another for four months, through 64 passages. In all instances the virulence of these strains of cold remained constant until they were discontinued.

The material for transmission of the above strains of cold was obtained from the nasal passages and sinuses of inoculated chickens that developed the disease. The chickens were sacrificed and the tissue and mucoid exudate within the palatine cleft, nasal passages and sinuses were scraped out and ground in a sterile mortar with sufficient 0.9 per cent sodium chlorid solution to form a 1:10 suspension. This was either centrifuged or passed through coarse filter paper to remove the tissue débris. This solution is referred to as the virus or virus solution.

TRANSMITTED COLDS

The disease was passed readily from one chicken to another by intranasal inoculation of the virus solutions. The chicken to be inoculated was wrapped in a sterile towel, with only its head protruding. The head was held first to one side and then to the

other, and the virus solution forced into each nostril four times by means of a small pipette, using about 2 cc of fluid altogether. To make certain that the virus solution passed beyond the intricate windings of the horny part of the nostrils, it was forced into the nostril until it ran out of the cleft palate. Then the beak was wiped dry and the chicken isolated in a sterile cage. Within 12 to 48 hours after inoculation, the chicken developed a watery nasal discharge which became more gelatinous and mucoid as the disease progressed. Most of the colds were accompanied by swellings under the eyes and some redness of the skin in this region. This swelling, however, receded within a few days and the eye assumed the normal appearance. The catarrh continued for several days, the duration depending upon the severity of the cold. Occasionally these symptoms were accompanied by the formation of a gelatinous exudate in the larynx, diagnosed from the mouth-breathing of the bird, but quite different from the gasping breath encountered in cases of laryngotracheitis, commonly known as the "gapes" (J. R. Beach,[3] Graham, Thorp and James[4] and Kernohan[5]).

Upon autopsy of a chicken suffering with a cold, all sinuses in the nose and eye region were found filled with the watery to gelatinous exudate. In the sinuses beneath the eyes there was often a soft yellow mass consisting chiefly of leucocytes. Occasionally there were pin-point extravasations of blood around the cleft palate and in the larynx. Spreads were made of the scrapings of the tissue around the sinuses, of the exudate, of the yellow masses and of the larynx tissue. These were stained by various methods, including Wright's blood stain, Giemsa's stain, hematoxylin and eosin, acid fuchsin and methyl green. The Wright's stain (Lewis and Gardner[6]) gave the most satisfactory results. Microscopic study of these spreads revealed no specific intracellular body, such as is common to most virus diseases, although numerous small granules were present in many of the cells and also scattered in the exudate. When the preparation was stained differentially to demonstrate the presence of mucus, these granules stained as mucous granules. Few bacteria were present in the early stages of the cold. No single type of organism predominated. The stained spreads showed mucus, white blood-cells, macrophages, epithelial cells and mucous-gland cells.

Incubation Period

All of the transmitted colds presented the symptoms described above and yielded a similar picture at autopsy. Of the 278 chickens inoculated intranasally with the untreated virus solution, 261

developed colds. In the chickens developing colds, the symptoms appeared in 161 within 24 hours after inoculation, in 72 within 48 hours, in 19 within 72 hours, and in the remaining nine within one week. In most instances the swollen eyes and nasal discharge appeared at approximately the same time. The swelling under the eyes remained for several days and then receded, while the nasal discharge continued for two or three weeks, sometimes disappearing for a day or two during this time. No definite change in rectal temperature occurred during the disease.

Attempts to produce the disease by intramuscular or by subcutaneous inoculation failed. The most rapid method of infection was found to be by direct transfer of a drop of the nasal discharge, withdrawn by means of a pipette from the upper nasal passages of a chicken suffering with a 24- to 48-hour cold, into the nasal passages of a normal chicken. Under these conditions symptoms of the cold usually appeared within six hours.

In 21 chickens inoculated with portions of the same virus solution on the same day and studied in order to follow the course of the disease, three died, 17 recovered within the usual time, and one still had the cold after 44 days. Seven of the chickens relapsed within eight to 20 days after apparent recovery and the second cold lasted two to six days.

Viability of Causative Agent of Chicken Common Cold

The cold-producing agent remained active in material preserved in glycerin and in dried material stored in an electric refrigerator. At varying intervals a portion of the preserved material was removed, the gelatinous exudate from the nasal and eye sinuses extracted as described above, and inoculated intranasally into other chickens. Storage for ten days in this manner neither decreased the virulence of the virus nor increased the length of the incubation period of the disease. However, after storage for 14 days, the incubation period lengthened and after 37 days the virus was no longer infectious.

Since in some of the experiments it was necessary to keep the chicken cold extract over a period of five or six hours, experiments were performed to determine how long and under what conditions the cold-producing substance would retain its virulence. A 1:10 extract of the exudate and tissue was found to remain infectious at room temperature for 36 hours; at 37° C. for 24 hours, and in the refrigerator for 48 hours, but failed to remain infectious when incubated for 48 hours. While a 1:100 saline extract remained active for eight to ten hours, it failed to produce a cold when kept in the refrigerator, at room temperature,

or in the incubator for as long as 48 hours. In several instances, 24-hour incubated broth cultures, prepared by adding about 0.5 cc of virus solution to 10 cc of sterile chicken infusion broth, brought about a cold within 48 hours after inoculation into the nasal passages of chickens. When the cold-producing agent remained active, its virulence remained undiminished and the incubation period was about the same as usual. From this it can be seen that the causative agent of the chicken cold readily resists destruction under ordinary laboratory conditions.

In order to determine the strength of the virus solution necessary to bring about a cold, dilutions of the chicken-cold material were made, using one gram of the original tissue as a basis. Colds of equal severity and equal incubation period resulted from dilutions ranging from one part of infectious tissue in 5 cc of normal salt solution to one part of tissue in 5,000 cc of saline. Various solutions were used as extractives in place of 0.9 per cent sodium chlorid. These consisted of Locke's, Tyrode's and Locke-Lewis solutions, chicken broth, beef broth, distilled water, hemolized blood, Dochez's medium and Kendall's medium. As none of these enabled the cold-producing agent to pass the bateriological filter, and as all of them resulted in an infectious virus solution, the sodium chlorid solution was used for routine purposes.

Susceptibility of the Virus Solution to the Action of Buffer Solutions

While the exudate taken directly from the nasal openings of a diseased chicken showed a hydrogen-ion concentration of 7.4 to 7.6, the virus solution usually was pH 6.8. As certain experiments involved treatment of the virus solution at different hydrogen-ion potentials, it became necessary to determine at what hydrogen-ion concentrations the virus solution would remain infectious.

Buffers ranging from pH 3 through pH 13 without preservatives were prepared by the LaMotte Chemical Products Company. These were used immediately upon delivery. To 5 cc of each buffer in sterile tubes was added 5 cc of a 1:10 virus solution and the tubes sealed with paraffined paper. The mixture was shaken well from time to time, the virus and buffer being permitted to stand in contact for ten minutes and then introduced intranasally into cold-free chickens. These experiments were repeated several times.

The cold-producing agent remained active in the mixtures of virus solution and buffers with hydrogen-ion concentrations from 4 to 12, but became inactive and failed to produce a cold in the

inoculated chickens when mixed with buffer solutions of pH 13. In more acid solutions the results were somewhat uncertain.

Alcohols at specific percentages have been used frequently to obtain enzymes from mixtures by precipitation of the enzyme. As this would be an excellent method for obtaining the cold virus in concentrated form, attempts were made to do so by using 50, 60 and 70 per cent alcoholic solutions of the virus. Chickens inoculated with the cloudy mixture of the 50 per cent alcohol solution of the virus developed typical colds. A control chicken inoculated with a 50 per cent alcohol solution, using broth in place of the virus, did not develop a cold. On another occasion the cloudy 50 per cent alcohol mixture of virus solution was centrifuged, after which the supernatant fluid was inoculated into one chicken, and the precipitate, after being washed well in sterile saline solution, and then suspended in fresh saline, was inoculated into another chicken. Both chickens developed colds. However, both the supernatant fluid and precipitate contained viable bacteria. Sixty and 70 per cent alcoholic suspensions were prepared and centrifuged as above, and the supernatant fluid and the washed precipitate of each were inoculated into separate chickens. No colds developed from the precipitates. However, as soon as the supernatant fluid was inoculated, there was swelling of the eyes and reddening around the nostril. Soon afterwards, a slight watery nasal discharge appeared. Both the swelling and the nasal discharge lasted for 24 hours and then disappeared. A control chicken, inoculated with 70 per cent alcohol alone, developed these same symptoms. Consequently, the cold was evidently not due to the virus but to the irritation of the alcohol itself.

The effect of a soap solution containing little free alkali was tested next. The soap solution was prepared by dissolving one gram of ivory soap in 100 cc of hot tap water and other dilutions were made from this, using hot tap water in each case. Chickens inoculated with virus treated with 0.05 per cent and 0.1 per cent soap solution for 20 minutes developed colds. Those inoculated with virus treated with 0.25, 0.50, and 1 per cent soap solution failed to develop colds.

Among disinfectants, mercuric bichlorid and formalin were tested. To 3 cc of a cold virus were added 3 cc of a 1:1,000 mercuric bichlorid solution and 3 cc of a 1 per cent formalin solution, respectively. After the virus and disinfectant had been shaken and allowed to stand for 15 minutes, each mixture was diluted by adding 2 cc of the virus-disinfectant mixture to 10

cc of sterile 0.9 per cent sodium chlorid. The resulting suspension was inoculated into chickens but no colds were produced.

The effect of thymol and toluol was observed. To 3 cc of toluol was added 3 cc of the cold virus, the mixture being shaken and allowed to stand for 15 minutes. The toluol layer was removed and the virus layer diluted (1:5) with saline, and then inoculated. No cold developed. When 10 mg of finely ground thymol was dissolved in 5 cc of the virus, and the mixture shaken, allowed to stand 15 minutes and then inoculated into chickens, no cold developed.

It was thought of interest to see whether the cold-producing agent could be inactivated by any of the dyes that had been found to inactivate certain filtrable viruses (Schultz and Kruger,[7] Frobisher,[8] Lewis and Lewis,[9] Clifton and Lawler[10]), enzymes and bacteria. Accordingly, to 5-cc portions of the virus solution were added various amounts of dyes. These were sealed with paraffined paper and shaken. The dye was left in contact with the virus solution for ten minutes, after which a portion of the solution was inoculated intranasally into a chicken and a few drops added to sterile chicken broth and incubated, to determine whether the growth of the bacteria present in the virus solution had been inhibited by the dye.

The dyes tested were methylene blue, toluidin blue, phenol indophenol, gentian violet, orange G, eosin, congo red, trypan blue and carmine. Gentian violet, methylene blue, toluidin blue and phenol indophenol, when added to the virus solution in amounts that inhibited the growth of bacteria, prevented the development of colds in chickens inoculated with such mixtures of dye and virus solution. The addition of smaller amounts of these dyes which did not inhibit the growth of organisms also sometimes inactivated the causative agent of the chicken cold, but the addition of amounts equivalent to 1:5,000 did not inactivate the cold-producing agent. Trypan blue, carmine, orange G, eosin and congo red did not inactivate the cold-producing agent, even when added in comparatively strong solutions. Neither did these dyes prevent the growth of organisms.

Filtrability

When chickens that had never been exposed to a cold were used to test the infectiousness of filtrates of the virus solution passed through the bacteriological filters, these filtrates almost never brought about a cold in the inoculated chicken, regardless of the type of filter used. However, when ordinary market chickens, supposedly free from colds, were used, the results were quite dif-

ferent. Almost 50 per cent of those inoculated with filtrate developed colds. This, as shown above, is about the percentage of market chickens that develop colds spontaneously.

During these investigations, covering a period of two years, filtration experiments were repeated again and again in the hope of finding some way to obtain a cold-producing filtrate. The filters used were the Berkefeld, with Mandler candle coarse, medium and fine, the Chamberland L_2 and L_3 and the Seitz. All filters used were absolutely clean, sterile and free from leaks. With the Seitz filter a new filter-pad was used for each filtration; the Chamberland bougie was burned out in an electric furnace and the Berkefeld filter was boiled in washing soda and washed in the usual way. The Seitz and Berkefeld filters were autoclaved after they had been cleaned and assembled, but with the type of Chamberland filter used no further sterilization was required. Filtration was carried out at 8 to 15 pounds pressure established by means of an electric vacuum pump apparatus. The hydrogen-ion concentration of the filtrates was determined and they were tested for bacteria. The control virus solution was taken from the unfiltered portion remaining in the filter when the filtration was concluded.

The results of the many filtrations were as follows: Of the 62 chickens inoculated with Seitz filtrates of 30 virus solutions, only three developed colds within seven days. Of the 70 chickens inoculated with Berkefeld filtrates of 34 virus solutions, two developed colds within seven days. Of the 60 chickens inoculated with Chamberland filtrates of 30 virus solutions, three developed colds within seven days, while all but three of the 85 chickens inoculated with the control virus solution taken from the filters developed colds within 24 to 48 hours.

Various alterations of the usual filtration procedure were tried in an effort to bring about the filtrability of the cold-producing agent. Some of these were as follows: filtering large quantities (100 to 500 cc) of the virus solution, dilution of the virus solution to be filtered, changing the hydrogen-ion concentration of the solution to be filtered, saturating the filter with a dye or with hemoglobin before filtering the virus solution, and adding soap, alcohol, hemolized blood or various dyes to portions of the virus solution to be filtered. None of these experiments, however, resulted in an infectious filtrate.

In view of the results obtained with the three types of bacteriological filters, an effort was made to prepare other types of

filters that might permit the active agent to pass. Thick filters were prepared of plaster of Paris (Kramer[11]), of barium sulfate, of bismuth subcarbonate and bismuth subnitrate each for x-ray, of animal charcoal, of aluminum hydroxid, and of calcined calcium sulfate. All of these filters, prepared of wet sterile particulate material, removed all or most of the proteids from the solution filtered, so that the resulting filtrates were water-clear and usually bacteria-free. While it was quite a simple matter to obtain a bacteria-free filtrate through this type of filter, the filtrate that proved to be free from bacteria did not bring about a cold, while those that proved to be contaminated, due to leaks or cracks, frequently produced a cold in the inoculated chicken. When thinner filters were prepared by using less particulate material, some filtrates were water-clear, others clear but slightly straw-colored and others slightly cloudy, depending upon the type and amount of particulate material used. These filtrates contained some proteids and most of them contained organisms. Those that were contaminated usually brought about colds in the inoculated chickens.

Adsorption

Following the unsuccessful attempts to filter an infectious cold-producing solution, adsorption experiments were undertaken in an effort to arrive at some explanation of this phenomenon (Lewis and Andervont[12]). Aluminum hydroxid, barium sulfate, bismuth subnitrate, bismuth subcarbonate, animal charcoal, commercial plaster of Paris, kaolin, kieselguhr, Fuller's earth, and calcined calcium sulfate were used as adsorbents.

An amount of adsorbent equivalent to a desired percentage was thoroughly ground with a chicken-cold virus and then removed by passing it through filter paper, all apparatus used having been previously sterilized. Some of the clear filtrate was inoculated intranasally into chickens and a portion of it added to broth and incubated in order to ascertain whether bacteria were present. The adsorption experiments were repeated as many as ten times, using amounts of adsorbent material equivalent to 5 to 25 per cent.

The results from the adsorption experiments, however, were most erratic. Sometimes adsorption with a larger amount of particulate substances resulted in a cold-producing filtrate, while that from a smaller amount of the same material did not bring about a cold in the inoculated chickens. On the whole, regardless of the type of particulate material or the amount of adsorbent used, the resulting solutions that proved to be free from

bacteria did not bring about a cold, while those that contained organisms produced a cold in the inoculated chickens.

Tissue Cultures

Although filtrates from the bacteriological filters failed to bring about a cold in the inoculated animals, it was hoped that the cultivation of filtrates of chicken cold in tissue cultures would permit the active agent to increase so that the inoculation of the tissue cultures would bring about the disease. Many types of tissue cultures, including hanging-drop cultures, dish cultures and rotary-tube cultures were tried. Flask cultures similar to those Rivers[13] found successful for the cultivation of vaccine virus were prepared and anaerobic tube cultures such as Dochez[14] described as favorable for carrying the human cold were prepared and transmitted through many subcultures. None of the cultures brought about the disease.

Immunity

At the time this work was in progress, it was impossible to carry out a study of the question of the immunity conferred upon animals recovering from colds owing to the difficulty encountered in keeping many chickens under suitable conditions. There seemed to be no doubt that following recovery from colds some chickens developed only a temporary and passing immunity while others became refractory to subsequent inoculations and remained so for a period of several weeks. Many of the chickens failed to recover from the cold and while their nasal passages were dry on some days, on others they were wet with nasal discharge. Some individuals developed a second cold when inoculated or when exposed to a chicken cold two weeks after recovery. Some were refractory to a second cold when inoculated four weeks after recovery. Practically all chickens that failed to develop colds following inoculation in a given experiment developed colds following a later inoculation, sometimes after having been refractory to one or two intervening inoculations.

Conclusion

The causative agent of the common cold of chickens failed to behave in a manner characteristic of the usual so-called filtrable virus diseases in that it did not confer a lasting immunity in the individuals recovered from the disease, it was not accompanied by a characteristic inclusion body in the cells of the lesion, it did not pass the bacteriologic filters, it was removed from solutions by amounts of particulate substances that removed bacteria,

and it was present in 24-hour incubated broth cultures of the virus, but not in those that were free from bacteria.

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A New Method of Branding

A painless method of branding cattle and horses is now being tried out in the West. The material used is a paint-like preparation, which burns through the hair and leaves a brand on the hide. The animal suffers no pain while the paint is being applied, an advantage over the old branding iron. While it is claimed that this type of brand does not injure the hide, that is a moot question, according to I. M. C. Anderson, live stock specialist, Montana State College, writing in the *Extension Animal Husbandman*. The paint-branding has been tried out with success on a number of animals used for experimental work in Montana.

Chappel Ranger

Joining the list of house organs published by makers of veterinary products is the *Chappel Ranger*, which recently made its appearance as the spokesman for Chappel Bros., Inc., of Rockford, Ill. The attractive four-page paper is done in rotogravure and is profusely illustrated. Any veterinarian who would like to receive this publication should write direct to Chappel Bros.

PARASITES COLLECTED FROM THE MOOSE, ALCES AMERICANUS, IN NORTHERN MINNESOTA*

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The purpose of this paper is to record, with annotations, the data accumulated on the animal parasites of moose, during the past three years, in the course of an investigation of the diseases of these animals, conducted by the staff of the Veterinary Division of the University of Minnesota, in coöperation with the Division of Entomology and Economic Zoölogy.[1]

This study has had a very definite practical angle because of the possibility that the so-called "moose disease" might be transferable to the cattle or other domesticated animals of the region. For this reason any data regarding the parasites, either external or internal, of moose may prove to be of significance.

Although a limited number of animal parasites have been reported from the European moose, *Alces alces*, there are few published records of those of our native species, *Alces americanus*. The increasing scarcity of these animals and the remoteness of their habitat from centers of scientific work have made them relatively unavailable for parasitological study.

From the 15 moose examined for parasites during the past three years, nine species of parasites have been taken. A list of these and their distribution in the host is given in table I.

EXTERNAL PARASITES

Dermacentor albipictus Packard, the winter or moose tick, was collected first on the moose in New Brunswick but since has been found on the horse, ox, beaver, elk, wapiti, mule-deer, white-tailed deer and mountain goat. Its geographic distribution extends across southern Canada and the northern United States from New Brunswick to the Rocky Mountains.

According to Bishopp and Wood,[2] the adults are found from February to April. The engorged females drop from the host in the late spring and early summer and oviposit on the ground. The eggs hatch during the summer and the larvae attach to the host in the fall. Both molts take place on the same host, the nymphs molting to the adult stage during the winter.

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Cameron[3] reports an outbreak of this tick on moose, horses and cattle in Saskatchewan. His observations on the life history agree essentially with those of Bishopp and Wood. This parasite appears to be universally present on the moose of Minnesota. Of the 15 animals examined, it was found on all but two. One of these was examined at a time (2-26-31) when the ticks are still in the nymphal stage and easily overlooked. The other was

TABLE I—*Parasites found in the examination of 15 moose in Minnesota.*

DATE	LOCALITY	SPECIMEN	AUTOPSIED BY	PARASITE*
2-26-31	Burntside Lake	Mature male, 4 years	C. R. Donham	4
3-16-31	—	—	R. Fenstermacher	2†
4-6-31	Winton, Minn.	Yearling male	R. O. Christenson	2, 7
5-9-31	Tofte, Minn.	Yearling female	R. O. Christenson	2, 9
4-23-31	Ensign Lake (Ely)	Yearling male	R. Fenstermacher and W. L. Jellison	1, 2, 3, 4
4-25-31	Ensign Lake (Ely)	Yearling female	R. Fenstermacher and W. L. Jellison	2, 3, 4
4-18-32	Cascade River on State Highway 1	Yearling male	R. Fenstermacher and F. G. Wallace	2, 3, 6, 7
4-30-32	Ely-Buyck Road	Mature female	R. Fenstermacher and E. F. Waller	2, 3, 4, 8, 9
5-1-32	Ely-Buyck Road	Mature male	R. Fenstermacher and E. F. Waller	2, 3, 4, 8, 9
6-2-32	Duluth, Minn.	Mature male	R. Fenstermacher and E. F. Waller	—
10-30-32	Lutsen, Minn.	Mature male	R. Fenstermacher and E. F. Waller	2, 3, 4, 9
12-5-32	Unknown—Confiscated	Yearling male	R. Fenstermacher and E. F. Waller	2†
2-17-33	Big Falls, Minn.	Yearling female	R. Fenstermacher, E. F. Waller and F. G. Wallace	2, 5
3-21-33	Mile 17, Gunflint Trail	Mature female	R. Fenstermacher and F. G. Wallace	1, 2, 3, 6, 7, 9
4-10-33	First Lake on Ely Buyck Road	Yearling male	R. Fenstermacher and F. G. Wallace	2, 3, 9

*Key: 1 = *Cysticerus* sp. (2); 2 = *Dermacentor albipictus* (13); 3 = *Dictyocaulus hadweni* (8); 4 = *Echinococcus granulosus* (6); 5 = *Fascioloides magna* (1); 6 = *Moniezia* sp. (2); 7 = *Nematodirella longispiculata* (3); 8 = *Paramphistomum cervi* (2); 9 = *Taenia hydatigena (Cysticercus tenuicollis)* (6). (Number in parentheses indicates total cases in which parasite was found.)

†No examination for 3 to 9.

examined on June 2, 1932, after the usual time for the adult ticks to leave the host and it showed unmistakable signs of having been heavily infested.

The thirteen other animals were heavily infested with ticks. Those examined in the fall and winter (10-30-32, 12-5-32 and 2-17-33) carried unengorged nymphs. The moose examined on March 21, 1933, harbored engorged nymphs, many just molting to the adult stage, and a few unengorged adults. The moose collected during the months of April and May were heavily infested with adult ticks in various stages of engorgement. Thus, in general, the seasonal distribution of these ticks on Minnesota moose agrees with the life cycle given by Bishopp and Wood.

On the moose the adult ticks are distributed over the entire body but are most numerous in the ears, between the fore and hind legs, on the belly and around the anus. On those areas which the animal can rub against trees, particularly the shoulders and belly, the hair is usually rubbed off and sore, scab-covered areas are left. It has been observed that where the ticks are most numerous the hair comes out very easily. These ticks are very numerous on the moose, in some places being so closely packed that there is no space between them.

Observations and experiments on the possible relation of this parasite to the deaths of moose will be published elsewhere.

NEMATODES

Dictyocaulus hadweni Chapin, a metastrongylid lung parasite, was described by Chapin[4] from specimens collected from the American bison. In addition to the type material, he examined specimens in the United States National Museum from *Alces americanus* and *Cervus canadensis*, and, although there were some slight differences between the parasites from the three hosts, they appeared to belong to the same species.

Dictyocaulus hadweni has been found in eight of the 15 moose examined. The parasites are found in the small bronchioles near the ends of the lobes of the lungs, where they occlude the air-passages and cause a marked congestion of large portions of the lung tissue.

Nematodirella longispiculata Yorke and Maplestone[5] was described by Romanovitch[6] under the name, *Microcephalus longissime spiculata* from the reindeer, *Tarandus rangifer*, in Russia. It has never been reported since, except for our records of its occurrence in the moose, where it has been found in three of the 15 specimens. The first specimens collected by R. O. Christenson were sent to G. Dikmans[7] for identification.

Nematodirella is found in the entire length of the small intestine and may be present in enormous numbers. The moose collected on March 21, 1933, was particularly heavily infected and showed some enteritis which might have been caused by the parasites.

Larval Tapeworms

The most common cestode in the moose is the larva of *Taenia hydatigena* Pallas or *Cysticercus tenuicollis*. This parasite was reported from the American moose by Liautard,[8] in 1880, and has since been recorded for the European moose by von Linstow.[9] It was found in six of the 15 Minnesota moose examined, in one case on the omentum and in all the others in the liver. In the specimen collected on March 21, 1933, there was a reasonably heavy infection, with twelve cysts in the liver. In addition to the normal cysticerci there were a number of small, white, fibrous cysts which might well have been degenerated tapeworm larvae. The experiments of Miller[10] on *Cysticercus fasciolaris* in the rat suggest that these may have been the result of superinfection.

R. O. Christenson identified the tapeworm larvae collected from the liver of the moose on May 9, 1931, as *Cysticercus tenuicollis*, the larval stage of *Taenia hydatigena*. This identification was experimentally confirmed by the author with other specimens.

Two cysts from the liver of the moose collected on October 30, 1932, were fed to a dog the next day. On March 31, 1933, or five months after infection, the dog was given 12 milligrams of arecolin hydrobromid and passed two mature tapeworms, 116 and 86 cm long, respectively, complete with their scolices. Another dog was fed four cysts from the moose examined on March 21, 1933. On May 8, 1933, or 49 days later, it was given a dose of areca nut and expelled three small tapeworms, 6.3, 5.6 and 1.3 cm long, respectively. Both dogs had been kept in the laboratory over a period of months and frequent fecal examinations had been made so the possibility of previous infection is very remote. Furthermore, one of the dogs had been fed nothing but commercial dog food since weaning.

Examination of the rostellum and the mature proglottids showed definitely that these tapeworms were *Taenia hydatigena*.

In six of the moose many-headed tapeworm cysts were found in the lungs. Those collected on February 26, 1931, were identified as *Echinococcus* by W. A. Riley. This identification was experimentally confirmed by the author at a later date when similar specimens were available.

In the moose collected on October 30, 1932, there were five of these cysts. One was reserved for morphological study and the

four others were fed on November 5 to a dog. On December 19, or 44 days after infection, the dog was killed and examined. The inner surface of the duodenum was practically covered with the adult *Echinococcus* (fig. 1) whose scolices were imbedded in the mucosa. These agree in every particular with the description of *Echinococcus granulosus* (Goeze) Rudolphi. The cystic stage of this tapeworm has been reported from a wide variety of mammals, including, *Alces alces*.

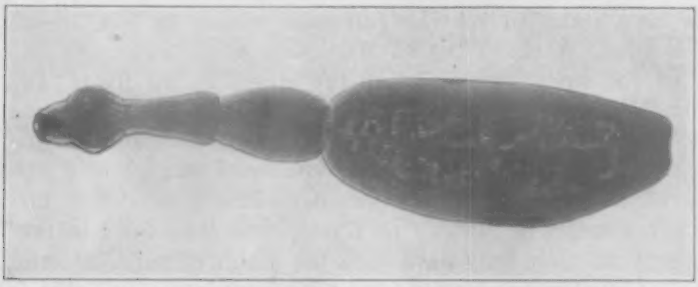

FIG. 1. *Echinococcus granulosus* reared in dog from cysts found in the lungs of moose (x 20).

Undetermined Cysticercus: In two of the moose (4-23-31) and (3-21-33) there were found imbedded in the heart cysticerci which strikingly resembled those of *Taenia solium (Cysticercus cellulosae)*. The agreement, both in size and in shape of hooks, was so close that if it were not for the host relationships the identification would not have been questioned. In the first animal there were five cysts approximately 1.5 cm long in the heart. Unfortunately, these were not critically studied. In the second moose were two cysts imbedded in the heart muscle, neither of which exceeded 0.5 cm in diameter.

ADULT CESTODES

In the intestines of two moose the author has found fragments of an adult tapeworm. Unfortunately, none of these specimens were in such condition that they could be specifically identified. However, they appear to belong to the genus *Moniezia*.

TREMATODES

Paramphistomum cervi Zeder has been collected twice. Specimens stained in borax carmine and cleared in oil of wintergreen showed the important taxonomic characters of the species. These parasites are found in large numbers attached to the wall of the rumen near the opening into the omasum. The species was reported from the moose by Diesing.[11]

Although *Fascioloides magna* Bassi has been reported many times from members of the *Cervidae* in Europe and America, it apparently has not been found previously in the moose. In one moose (2-17-33) there were 13 specimens (fig. 2) in the liver. The largest measured 27 mm in length and 14 mm in width, while the smallest was 7 mm in length and 4 mm in width. The average size was 13.9 mm x 7 mm. This is much smaller than the *Fascioloides* ordinarily found in deer and cattle but nevertheless the morphological features seem to be identical with those of the larger specimens. Furthermore, Francis[12] reports that he found flukes of this species as small as 8 mm x 4 mm in cattle in Texas.

FIG. 2. *Fascioloides magna* from liver of moose (x 3).

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A NOTE ON SO-CALLED QUAIL DISEASE*

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The so-called "quail disease" to be described herein appeared suddenly, during the summer of 1932, on a quail farm that had been in operation less than nine months. The sandy, well-drained soil used for breeding-pens had been in cultivation for 50 years and no domestic fowl, as far as could be learned, had been reared on the same ground. Quail were hatched in electric incubators and kept in screened sanitary brooding-pens to exclude flies and mosquitoes. (See figure 1.) The young quail were kept continuously on wire, to avoid soil contamination, until eight or ten weeks of age, and then on the ground in portable range pens, which were moved to fresh ground each week.

FIG. 1. Section of combination pens in foreground. Combination pens can be used either as laying-pens or growing-pens. Midsection of photograph shows holding-pens 50′ x 10′ x 6¼′ high. At extreme right, elevated growing-pens.

Preceding the acute losses referred to as quail disease, coccidiosis developed in the breeding stock during the latter part of the breeding season and approximately 18 out of 140 birds succumbed, presumably to this disease. Autopsies of typically affected breeders showed an occasional, slight, hepatic blackhead involvement. As the losses in the breeding flock subsided, the onset of a fatal disease in poults was experienced. The disease in young quail

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developed spontaneously in 100 of the 150 pens. Only 50 of 150 pens escaped the disease. Each pen had about 14 young quail. Some pens were entirely wiped out; in others over one-half of the quail died, and in still other pens only one or two birds survived. Out of approximately 2,000 young quail placed in range pens, more than 1,400 succumbed.

Similar acute losses, associated with a variety of bacteria, have been reported independently by Morse and Gallagher[1] in non-domesticated birds. These losses were described as due to an infectious disease of the grouse family caused by a microbe of the *Bacterium coli* group. This microörganism is present normally in the intestine and is non-pathogenic under ordinary conditions. When, however, birds are subjected to unusual conditions associated with shipping, overcrowding with lack of sanitary precautions, or other unhealthful influences, the resistance of the body may be lowered to such an extent that the germ can attack the weakened organs and cause serious trouble. Gallagher, however, expressed doubt that *Bacterium coli* was always responsible for the condition known as "quail disease" and suggested that coccidia might bear a causative relation to the lesions. His experiments showed that quail under normal conditions were resistant to this organism although he commented:

> It undoubtedly does considerable damage as a secondary invader and may possibly initiate the disease under certain conditions.

Shillinger, Pickens and DeVolt,[2] in 1932, isolated "colon" and paratyphoid organisms from tissues of bob-white quail dying of a so-called "quail disease" on the Maryland State Game Farm. The isolated organisms, when injected directly into the body cavities of guinea pigs, produced death in the majority of cases. Quail injected with the organisms died in two days or less. However, one quail, fed a culture of the colon organism, remained apparently healthy for two months following the feeding. The results suggest a combination of lowered resistance of the birds and a bacillary invasion of colon, paratyphoid or other organisms as secondary causative agents.

Examination of Quail

During a period of two months (1933), ten quail were received at the Laboratory of Animal Pathology and Hygiene for autopsy and examination. The specimens were iced for shipment and arrived at the laboratory in excellent condition. Direct cultures were made from the heart-blood, liver and spleen of each bird on plain and blood-agar plates, and portions of these tissues were incubated in plain broth and plain agar shakes. The direct cul-

tures were sterile, in most instances, after incubation for two to four days at 37° C., although gamma and alpha streptococci, a diplococcus, a small Gram-negative cocco-bacillus and a small Gram-negative rod were isolated occasionally.

A suspension was made in physiological salt solution, of the heart, liver and spleen tissues of one group of two quail received. Direct cultures from this lot were negative. One portion of the emulsion was injected subcutaneously in 1-cc amounts into a rabbit, guinea pig, two pigeons, and two young chickens. Duplicate animals received similar amounts of a bacteriologically sterile filtrate prepared by passing the emulsion through a Berkefeld N filter. One of the chickens inoculated with unfiltered material died in four days and a diplobacillus was isolated from direct culture. The guinea pig injected with unfiltered material died in 17 days and direct aerobic cultures on plain and blood agar were negative.

The rabbit injected with the same material died in 18 days and a culture of a paratyphoid-like microörganism was isolated from the heart-blood.* The remaining chicken died in three weeks. Cultures were negative. The pigeons were released as healthy, as were the animals receiving the filtrate, two and one-half months after injection. Tissue emulsion and filtrate from another quail were each injected subcutaneously as follows: Rabbit, 2 cc; guinea pig, 1 cc; pigeon, 1 cc; quail, 0.5 cc. The quail and pigeon receiving the emulsion died in 24 hours; the rabbit died in 20 days. A large Gram-negative rod, as well as *E. coli*, were isolated from the pigeon. The guinea pig, as well as the animals receiving the filtrate, were released as healthy in 20 days.

Two tissue emulsions (1 and 2) from the other quail examined were injected subcutaneously as follows: 2 pigeons, 2 cc each; 2 chickens, 1 cc each. The pigeons died within 24 hours after injection and showed lesions suggestive of septicemia, with bloody imbibition at the point of injection. Cultures yielded an alpha streptococcus. The chickens were released as healthy in two weeks. One cc of the emulsion was given subcutaneously to a guinea pig and pigeon, and 2 cc to a rabbit. The rabbit died in four days and the guinea pig died in two days, while the pigeon

*Autopsy revealed an enlarged spleen, and grayish circumscribed areas on the liver; smears were negative to coccidiosis. Multiple abscesses were found in the liver; also in lymph-glands and mesentery. The liver was emulsified in physiological salt solution and injected subcutaneously in 2-cc and 1-cc amounts into a rabbit, a guinea pig and a pigeon. The rabbit succumbed in 17 days and the guinea pig in 14 days. Paratyphoid cultures were isolated from each by direct culture. The pigeon was released healthy after 21 days. In 1924, Beaudette and Edwards[4] isolated *S. aertrycke* from an epizoötic outbreak among canary birds kept in captivity. Emmel and Stafseth[5] also isolated *S. aertrycke* from canary birds dying in five outbreaks of an epizoötic occurring in bird-stores throughout the state of Michigan.

was released as healthy in one month. A gamma streptococcus was recovered from the spleen of the guinea pig.

Autopsy Findings

Most of the quail received for examination were fat and in good condition, only a few being thin and emaciated. The following gross autopsy findings are somewhat typical:

Liver: Very soft, congested; color varies from light, with grayish areas, to normal; or very dark with necrotic areas.

Heart: Usually firm and normal; may show vessels highly congested or be covered with urates.

Lungs: Congested; no pneumonia.

Spleen: Normal.

Kidneys: Usually normal; may be enlarged and light in color, or very dark.

Proventriculus: Usually normal; may show catarrhal inflammation.

Gizzard: Normal.

Intestine: Congested; much catarrhal, mucoid material; mild enteritis; no coccidia present.

Examination of Quail Eggs

Fifty-six quail eggs were examined for evidence of bacterial contamination. The eggs were immersed in a 1:1,000 solution of bichlorid of mercury, the tip washed with alcohol, and daubed with tincture of iodin preparatory to puncture of the shell. Samples were taken with sterile capillary pipettes and cultured on plain agar plates and in plain broth. The eggs then were composited in physiological salt solution and the emulsion was injected subcutaneously in 1-cc amounts into a guinea pig and a pigeon, and 2 cc given a rabbit. The inoculated animals remained apparently healthy for 19 days, at which time they were killed and cultures from the heart-blood, liver and spleen were made on plain agar plates. After five days at 37° C., the inoculated plates remained negative.

Pathogenicity of Bacteria Isolated

Five types of organisms were isolated by direct cultures from the heart-blood, liver and spleen of the quail, while six isolations were made from animals injected with tissue emulsions. All cultures were tested for pathogenicity.

Direct isolation: Alpha and gamma streptococci, washed off agar slants with physiological salt solution, and injected subcutaneously in 1-cc amounts, failed to produce illness or death in

guinea pigs or pigeons in two weeks to one month. A rabbit injected subcutaneously with 1 cc of a culture of a diplococcus was released as healthy in 15 days. Two pigeons injected with 1 cc of a culture of a Gram-negative coccobacillus were released as healthy in 15 days. A pigeon injected with 1 cc of a culture of a small Gram-negative rod died in four days. A gamma streptococcus was isolated but did not prove pathogenic for four pigeons when injected in 1-cc amounts.

Animal isolation: Diplobacillus (through chick): Two pigeons fed the contents of one agar slant, washed off in physiological salt solution, daily for ten days, were released as healthy in one month. *E. coli* and a large Gram-negative rod (through pigeon): Two pigeons injected with 1-cc amounts of these cultures were released as healthy in 15 days. Alpha streptococci (through pigeon): Pigeons injected with 1-cc amounts of this culture were released as healthy in three weeks. Gamma streptococci (through guinea pig): The contents of one slant, washed off in physiological salt solution, fed daily for ten consecutive days, did not produce illness or death in two pigeons. Paratyphoid (through rabbit): A pigeon injected with 1 cc of a physiological salt solution of this organism died in six days and a paratyphoid organism was isolated from the heart-blood, liver and spleen. Two pigeons, however, fed the contents of one agar slant daily for ten consecutive days, were released healthy in one month. The culture in similar amounts was fed also to four bob-white quail that remained healthy for 21 days. One quail injected subcutaneously with 0.5 cc died in two days. Paratyphoid was isolated. Autopsy revealed congestion of the liver, lungs and intestine. No coccidia were present.

IDENTIFICATION OF S. AERTRYCKE

The above data suggest the pathogenic character of the paratyphoid strain isolated. An attempt was made to classify this organism by cultural as well as by morphological and serological methods. Strains of the organism showed it to be a short, Gram-negative rod with neither spores nor capsules. The growth on agar slants and agar plates was abundant and resembled that of the colon-typhoid group. Litmus milk became slightly acid upon inoculation but became alkaline in 24 to 48 hours. Plain broth became cloudy with a viscous sediment in 48 hours. Indol was not formed, while nitrates were reduced to nitrites.

The organism was agglutinated with para A, para B, para C, typhosus and pullorum immune sera (table I). Cultures of *S. aertrycke and S. pullorum* were run as controls. Dilutions of

TABLE I—*Agglutination reactions.*

CULTURE	AGGLUTINATION REACTIONS																					
	PARA A SERUM*					C	PARA B SERUM					C	PARA C SERUM					C	TYPHOSUS SERUM					C	S. PULLORUM SERUM					C
Organism I	±	−	−	−	−	−	+	+	+	±	−	−	−	−	−	−	−	−	+	+	+	±	−	−	+	+	+	+	±	−	−	−
Organism II	±	−	−	−	−	−	+	+	+	+	−	−	−	±	−	−	−	−	−	+	+	−	−	−	−	+	+	+	±	−	−	−
Organism III	−	−	−	−	−	−	+	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+	±	−	−	−
S. aertrycke	−	−	−	−	−	−	+	+	+	−	−	−	−	±	−	−	−	−	−	+	−	−	−	−	−	+	+	+	−	−	−	−
S. pullorum	+	+	−	−	−	−	+	+	−	−	−	−	−	−	−	−	−	−	−	+	+	+	+	−	−	+	+	+	+	−	−	−

*Dilutions: 1:25, 1:50, 1:100, 1:200, 1:400, 1:800, 1:1600, 1:3200. C = Control.

Source of cultures:

Organism I, isolated from heart-blood of rabbit injected with quail tissue.
Organism II, isolated from heart-blood of pigeon injected with organism I.
Organism III, isolated from liver of quail injected with organism II.
S. aertrycke, A. M. S. (Mutton) 41-H-2 L. No. 120 B. A. I.
S. pullorum, 4726.

1:25, 1:50, 1:100, 1:200, 1:400, 1:800, 1:1,600 and 1:3,200 were employed. While the organism was agglutinated in a 1:200 serum-antigen dilution with para B serum and gave no trace of agglutination with para A and para C sera, it was agglutinated in a dilution of 1:400 by the *S. pullorum* serum. The control *S. pullorum* culture, however, was agglutinated by the *S. pullorum* serum in a 1:1,600 dilution. The organism behaved in these agglutination tests in the same manner as the *S. aertrycke* control. Agglutinin-absorption tests were not run.

Acid and gas in dextrose, maltose, xylose, arabinose and inositol were produced. No acid or gas was produced in lactose, sucrose or dextrin (table II). The production of acid and gas in dextrose and maltose, with no acid or gas in lactose and sucrose, confirmed the paratyphoid classification. The organism was planted also on differential media[3] and gave an acid reaction, differentiating it from *B. paratyphosus A and B. schottmülleri* which give alkaline reactions. The fermentation of xylose and inositol indicated the organism was not *B. paratyphosus A*. The fermentation of arabinose and inositol excluded identity with *S. suipestifer*. *B. enteritidis* also fails to ferment inositol.

TABLE II—*Carbohydrate reactions.*

	Differential Media	Carbohydrate Reactions							
		Dextrose	Lactose	Maltose	Sucrose	Xylose	Arabinose	Inositol	Dextrin
Organism I	+	++	−	++	−	++	++	++	−
Organism II	+	++	−	++	−	++	++	++	−
Organism III	+	++	−	++	−	++	++	++	−
S. aertrycke	+	++	−	++	−	++	++	++	−
S. anatum	+	++	−	++	−	++	++	−	+
S. pullorum	−	++	−	−	−	++	++	−	−
S. schottmulleri	−	++	−	++	−	++	++	++	++

Source of cultures:

Organisms I, II and III, *S. aertrycke* and *S. pullorum*, see table I. *S. anatum*, 17008. *S. schottmülleri*, A. M. S. (Newport) 41-H-1 L. No. 121 B. A. I.

SUMMARY

1. The presence of a filtrable virus was not demonstrated in tissues of fatally affected quail.

2. Streptococci, diplococci and *E. coli* were isolated in direct culture from quail tissues. *S. aertrycke* was isolated from one spontaneously fatally affected quail *via* inoculation of tissue emulsion into susceptible animals.

3. Bacteriologic examination and animal inoculation of 56 quail eggs from the breeding flock failed to yield any evidence of the presence of a pathogenic virus.

4. The immeasurable environmental factor or factors, such as confinement *per se*, as well as hatching, rearing and feeding quail on restricted range, might obviously culminate in disturbed or altered physiologic functions. In such quail it is reasonable to assume that non-pathogenic agents might assume a pathogenic rôle.

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Bob Becker Broadcasts Dog Stories

"Writing a first-class dog program is no easy matter," said Zipper, who in private life is Bob Becker's lively wire-haired terrier. "For there are two ways of looking at dog life—from the master's side, and from the dog's side. Now this master of mine, Bob Becker, is a pretty grand fellow. He lets me go over every part of his radio yarns and dog stories to be sure they're just exactly right from the dog's point of view."

Bob Becker, who is well known through the many worlds of dogdom, exploration and hunting, has gathered around him an enthusiastic audience of dog lovers and those who love the out-of-doors. Twice a week over WGN, Chicago; WBZ, Boston; KMOX, Saint Louis, and KSTP, Saint Paul, he broadcasts these interesting chats on dogs. Covering not only their care and handling, Becker gives stories from his own varied experiences with dogs from the Arctic Circle to Brazil. Recently he interviewed Dr. John W. Patton (Tex. '21), of East Lansing, Mich., on the feeding of dogs.

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EQUINE ENCEPHALOMYELITIS IMMUNIZATION*

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The prevalence of equine encephalomyelitis in the western states from 1930 to date has prompted active investigation into the various aspects of this disease by several research organizations. Detailed accounts of the clinical symptoms, anatomical changes, epidemiology and the nature of the virus involved have been reported by several writers.

In 1933, a disease of equines appeared in epizoötic form in several eastern states with practically identical clinical and epidemiological manifestations. It has been found, however, that the filtrable virus involved is not immunologically and serologically related to the western strains.

In 1933, we published a method for the quantity production of antiviral serum effective against the western type of virus,[1] as well as a general outline of the treatment and control of the disease based on the use of this serum.[2] However, it was realized that antiviral serum had distinct limitations in a disease of this nature and some more effective means of prevention should be sought. Due to lack of definite information regarding the methods of transmission and routes of infection under natural conditions, a method of effective and enduring immunization appeared to offer the only practical means of controlling epizoötics.

Extended experimental work at this station, not covered in this report, thus far has failed to develop a method of modifying or attenuating equine encephalomyelitis virus without also destroying its immunizing properties. As soon as the power to infect guinea pigs by the intracranial route was lost, the original virus-bearing material was nonantigenic. Work along this line is being continued, however, as a modified virus entirely incapable of producing the disease by any method of injection but still antigenic would be the ideal immunizing agent.

Early in our work with this disease, it was observed that the several Nevada strains of virus with which we were working possessed one common peculiarity. They practically never produced fatal infection in guinea pigs or horses when administered by skin scarification, intradermal or subcutaneous injection, regardless of dosage or virulence of the virus used. At most only mild reactions such as a moderate temperature rise resulted. It

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was found also that such virus administration produced a solid immunity to intracranial injections of highly virulent virus in practically every instance.

Preliminary Field Trial on Horses

An opportunity to apply this observation in the field presented itself in 1932. In July of that year, encephalomyelitis appeared in a group of about 55 horses on one of our state institution ranches. A total of eleven cases occurred within a period of about six weeks. With the consent and coöperation of those in charge, it was decided to attempt the active immunization of these horses.

On August 18, 41 head of these horses ranging in age from four months to 21 years were given their first virus injection. The material used was a 2 per cent suspension in Locke's solution of brain tissue from a horse killed nine days previously in an advanced stage of encephalomyelitis following intracranial inoculation. The material received no treatment other than grinding aseptically in a sterile mortar with sterile sand and straining through gauze. This virus suspension was not titrated to determine the definite m. l. d. for guinea pigs but a 0.2-cc dose intracranially killed 400-gram guinea pigs with typical encephalomyelitis in six days. The dosage used on the horses was 2 cc for colts up to six months of age and 5 cc for the older horses.

With one exception, no rise of temperature or other noticeable symptoms followed the injection. Nine days after the first virus injection, a seven-year-old gelding showed definite symptoms of encephalomyelitis but fully recovered in four days under serum treatment. As the disease was active on this ranch at the time of injection, there was no way of definitely determining whether this case was induced by the virus injection, a natural infection or a combination of both. This was the last case to appear.

On September 1, twelve days after the first injection, these horses were given a second injection of virus. This material was prepared in a practically identical manner but the dose was increased to 4 cc for the colts and 10 cc for the balance of the group. No rise of temperature or other departures from normal followed this second injection. Control guinea pigs injected intracranially with this same lot of virus suspension developed typical encephalomyelitis symptoms and died.

As far as deriving definite conclusions was concerned, this experiment was open to obvious objections. As encephalomyelitis had been present for at least six weeks, the influence of recent naturally acquired immunity on the results could not be deter-

mined. Adequate controls were not provided although three contact horses not treated for various reasons did not develop the disease. The results did at least indicate that the procedure followed did not increase the incidence of the disease in a group of horses where it was already active and apparently completely suppressed it within ten days.

First Controlled Experiment on Horses

In the spring of 1933, it was decided to try this method of immunization on horses under strictly controlled conditions. For this purpose a group of 20 horses and two mules were collected from the isolated ranges of northwestern Nevada where encephalomyelitis had never been reported. The ages ranged from one to 15 years and mares and geldings were included.

The animals were divided into three groups. Group I, consisting of seven head, received a single subcutaneous injection of brain virus. Group II, also consisting of seven head, received two injections of brain virus at intervals of 14 days. Group III, consisting of eight head, were left as untreated controls.

On May 5, 1933, the horses in group I were injected subcutaneously with 10 cc of a 2 per cent suspension of virus A in Locke's solution. On the same date, the group II horses received 5 cc of the same suspension. This A strain of virus had been maintained for 18 months by frequent passage through guinea pigs and horses and its behavior under laboratory conditions was well known. It was moderately virulent but never had caused infection in horses even when 50 cc of a 2 per cent brain suspension was injected subcutaneously, although it consistently caused infection in susceptible horses when injected intracranially in 3- to 5-cc doses.

The virus suspension used was held for two days at 5° C. after preparation. Titration on the day when the horses were injected showed an m. l. d. of 0.2 cc of a 1:800 dilution for 400-gram guinea pigs upon intracranial injection. This brain virus suspension proved to be free of bacteria on cultural test.

Fourteen days later, on May 19, the group II horses were given a second subcutaneous injection of 10 cc of a 2 per cent Locke's solution suspension of virus B. This virus was unusually virulent for guinea pigs and horses upon intracranial injection, but had not heretofore produced symptoms in horses or guinea pigs when injected subcutaneously.

We had previously determined by cross-immunity tests on guinea pigs that virus A and virus B were immunologically identical, but virus B was a more active virus. The 2 per cent Locke's

solution suspension was prepared with unmodified guinea pig brain-tissue virus and held at 5° C. for one day prior to use on the horses.

Titration of this virus suspension showed 0.2 cc of a 1:25,000 dilution to be the m. l. d. for 400-gram guinea pigs. The group II horses therefore received approximately 25,000 guinea pig m. l. d. at the second injection. All injections were made subcutaneously and as close to the skin as possible, in some instances the injection actually being partially intradermal. Details of the treatment of these three groups of horses and the results are shown in table I.

The three groups of horses summarized in table I were kept together in the same corral and observed daily. Post-injection temperatures of the more tractable individuals from all three groups were taken daily. There was no rise in temperature nor other unfavorable symptoms. The control horses were placed in contact with the virus-injected horses purposely, to test out the possibility of virus carriers or spreaders among the injected horses causing spontaneous cases of the disease among the controls.

No spontaneous cases occurred among the controls during seven months of contact with virus-injected horses. Furthermore, it appears that the controls did not acquire any natural resistance that could be detected either by the virus neutralization test on their serum *in vitro* or immunity to intracranial injection of virus. All but one control proved susceptible to either intranasal or intracranial injection of virus after from one to seven months of exposure to the vaccinated horses.

From preliminary immunity observations on guinea pigs following intradermal or subcutaneous injection or the application of virus to the scarified skin, it was found that solid immunity to intracranial virus injection was not uniformly present until about 21 days after such treatment, at which time they would resist massive doses of extremely virulent virus injected intracranially. As a result of this observation on guinea pigs, the first immunity test was made on four horses from group I, 20 days after subcutaneous injection with a single dose of virus. At the same time two controls also were inoculated. Two of the vaccinated horses and one control received 5 cc of a 2 per cent suspension of virus B intracranially under anesthesia*; two vaccinated and one control received 50 cc of the same suspension instilled into the nostril daily on three consecutive days.

*All intracranial injections on experiment animals were made under general anesthesia. Pentobarbital sodium was used on the horses and ether on guinea pigs.

The four horses from the vaccinated group survived without symptoms. The two controls developed typical symptoms of encephalomyelitis in four and seven days, respectively. Both controls became prostrate and were destroyed when moribund.

TABLE I—*Virus-alone immunization.*

ANIMAL			IMMUNIZATION		IMMUNITY TEST			RESULT OF IMMUNITY TEST
HORSE	AGE (YRS.)	SEX	5-5-33 VIRUS A*(cc)	5-19-33 VIRUS B* (cc)	DATE (1933)	VIRUS DOSE* (cc)	METHOD †	
8348	1	F	10	— — —	5-25	5	CRA	No reaction
8349	12	F	10	— — —	5-25	5	CRA	No reaction
8350	5	F	10	— — —	5-25	50	NAS	No reaction
					5-26	50		
					5-27	50		
8351	20	M	10	— — —	5-25	50	NAS	No reaction
					5-26	50		
					5-27	50		
8353	1	F	10	— — —	6-23	5	CRA	No reaction
8357	20	F‡	10	— — —	8-4	60	NAS	No reaction
8354	7	M	10	— — —	10-20	5	CRA	D. 10-27-33 (E)§
8358	18	F	5	10	6-2	5	CRA	No reaction
8365	10	M‡	5	10	6-2	5	CRA	No reaction
8360	9	M	5	10	6-2	50	NAS	No reaction
					6-3	30		
					6-4	50		
8363	8	M	5	10	6-2	25	NAS	No reaction
					6-3	30		
					6-4	50		
8366	20	M	5	10	6-23	50	NAS	No reaction
					6-24	50		
					6-25	50		
8359	4	F	5	10	8-4	60	NAS	No reaction
8361	2	F	5	10	10-20	5	CRA	D. 10-26-33 (E)
8371	2	F			5-25	5	CRA	D. 5-30-33 (E)
8377	11	M			5-25	50	NAS	D. 6-3-33 (E)
					5-26	50		
					5-27	50		
8370	2	F			6-2	5	CRA	D. 6-8-33 (E)
8372	15	M			6-2	50	NAS	D. 6-11-33 (E)
					6-3	25		
			Controls		6-4	30		
8379	1	F			6-23	5	CRA	D. 6-29-33 (E)
8375	1	F			8-3	60	NAS	No reaction
					8-17	100		
8373	28	F			10-20	5	CRA	D. 10-25-33 (E)
8374	2	F			10-20	5	CRA	D. 10-26-33 (E)

*Two per cent brain-tissue virus suspension used.
†CRA = Intracranial injection under anesthesia.
NAS = Instillation into upper portions of nasal passages.
‡Mule.
§(E) = Encephalomyelitis.

The second series of immunity tests were made using four horses from group II which had received two subcutaneous injections of virus, 5 cc of virus A and 10 cc of virus B, at intervals of 14 days, two weeks after the last injection. Two of the group II horses received 5 cc of a 2 per cent brain suspension of virus B intracranially. Two received 50 cc of the same suspension instilled intranasally, without traumatism, daily on three consecutive days. One control received 5 cc of the same virus suspension intracranially and one was given 50 cc intranasally daily on three consecutive days.

All of the group II horses remained symptom-free. The controls developed typical encephalomyelitis symptoms, became prostrate and were destroyed when moribund. The control which received virus in the nostril showed initial symptoms on the fifth day and was destroyed on the ninth day. The control which received virus intracranially showed locomotor disturbances on the fifth day following virus injection and was destroyed on the sixth day when moribund.

From the first series of immunity tests it was obvious that a single subcutaneous injection of 10 cc of a moderately active virus afforded complete protection against an intracranial dose of very active virus administered 20 days later.

The remainder of the virus-injected horses and untreated controls were periodically exposed to virus, either by the intracranial or intranasal methods, from 20 days after first vaccination until six months had elapsed.

Horse 8350, which resisted intranasal virus instillation on May 25, 26 and 27, was purposely retained, as she foaled during the test period. When tested by intracranial injection of virus six months later, she developed typical encephalomyelitis symptoms and was destroyed.

Horses 8354 and 8361 were given their first immunity test five and one-half months after virus vaccination. Both of them developed severe symptoms and paralysis and died six and seven days later. This would indicate that the immunity had decreased during the five and one-half months to a level which would no longer resist intracranial injection of the standard dose of virus.

As an additional control on horse inoculations with virus at each periodic immunity test, at least three guinea pigs were injected intracranially with the same virus suspension. In every instance all of the guinea pigs developed typical symptoms of virus infection and either died or were destroyed when moribund, thus demonstrating that the virus suspensions were active in every instance.

It is recognized that intracranial injection of virus is a very severe test of immunity and greatly in excess of any exposure likely to occur under natural conditions. In our experience six out of ten (60 per cent) presumably susceptible horses developed typical symptoms of encephalomyelitis following the intranasal instillation of virus. The possibility that intranasal instillation of virus may actually increase the resistance of horses already partly immune to this virus may account for the neutralization of virus by the serum of horse 8359, as well as resistance to intracranial injection of virus six months after immunization.

The foregoing experiment indicated that the subcutaneous injection of rather large amounts of unattenuated virus into susceptible horses was harmless and produced a solid and fairly lasting immunity.

Second Controlled Experiment on Horses

It was decided next to ascertain the effect of even larger doses of virus alone, as well as graduated doses of virus given simultaneously with a constant dose of antiserum. Another group of 14 horses and one mule was secured from the same region for this purpose. Ages ranged from one to ten years; mares predominated. A group of six were given virus alone in variable doses. Another group of six received the same dosages of virus and 50 cc of antiserum each. Three were left untreated to serve as controls.

Virus B was used on these horses in the form of brain tissue from two horses, ground to a 20 per cent suspension in Locke's solution, held six days at 5° C. and diluted to 2 per cent at the time of use. Titration on the day the horses were injected showed an m. l. d. of 0.2 cc of a 1:25,000 dilution of this brain-tissue suspension for 400-gram guinea pigs. Three doses of virus were used, namely 5, 10 and 20 cc, representing approximately 12,500, 25,000 and 50,000 guinea pig m. l. d., respectively. The antiserum used was a highly potent lot prepared and tested as previously described.[1] Details of the treatment of these horses and the results are shown in table II.

These groups of horses were kept together under close observation and temperatures taken daily following injection. One two-year-old mare (8541), which received a 10-cc dose of virus alone, developed peracute symptoms of encephalomyelitis and paralysis on the tenth day after injection and was destroyed late on the same day. Virus was recaptured from the brain. The other injected horses remained symptom-free until the time of the immunity tests.

In order to determine whether any immunity was present on the 14th day, six vaccinated horses and one control were each injected intracranially with 5 cc of a 2 per cent brain-tissue suspension of virus B. Four of the six horses showed mild to severe symptoms of encephalomyelitis as a result of the intracranial injection of virus but recovered without apparent disability. The control injected intracranially with virus on the same date developed typical symptoms and prostration and was destroyed when moribund.

Twenty-two days after vaccination, the other six vaccinated horses and two controls were injected intracranially with 5 cc of a 2 per cent brain suspension of virus A. One vaccinated horse

TABLE II—*Simultaneous virus-serum immunization.*

ANIMAL			IMMUNIZATION		IMMUNITY TEST			
HORSE	AGE (YRS.)	SEX	7-20-33 VIRUS (CC)	7-20-33 SERUM (CC)	DATE (1933)	VIRUS DOSE* (CC)	METHOD †	RESULT OF IMMUNITY TEST
8538	2	F	5	0	8-4	5	CRA	No reaction
8539	1	F	5	0	8-11	5	CRA	No reaction
8540	1	M	10	0	8-4	5	CRA	Severe reaction. Recovered
8541	2	M	10	0	.....	.....	.....	K. 7-30-33 (E)‡
8542	1	F	20	0	8-4	5	CRA	Mild reaction. Recovered
8543	10	F	20	0	8-11	5	CRA	No reaction
					8-17	50	NAS	No reaction
8544	8	F	5	50	8-4	5	CRA	No reaction
8545	4	F	5	50	8-11	5	CRA	No reaction
8546	2	F	10	50	8-4	5	CRA	Mild reaction. Recovered
8547	1	M	10	50	8-11	5	CRA	No reaction
					8-17	50	NAS	No reaction
8548	7	F	20	50	8-4	5	CRA	Rather severe reaction. Recovered
8549	7	F	20	50	8-11	5	CRA	Rather severe reaction. Recovered
8552	3	F	Controls		8-4	5	CRA	K. 8-11-33 (E)
8551	6	F			8-11	60	NAS	No reaction
					8-17	60		No reaction
8553	2	M			8-11	5	CRA	No reaction
					8-17	60	NAS	No reaction

*Two per cent brain-tissue virus suspension used.
†CRA = Intracranial injection under anesthesia.
NAS = Instillation into upper portions of nasal passages.
‡(E) = Encephalomyelitis.

(8549), developed mild symptoms without paralysis and fully recovered in ten days without treatment.

Neither of the controls developed symptoms as a result of the virus injection. A superimposed infection by nasal instillation also was attempted on four of these horses seven days later without any visible disturbance.

Apparently the controls had acquired some natural resistance either prior to entrance on the experiment or during the interval of three weeks in contact with the injected horses, some of which showed symptoms following the first immunity test.

There is no doubt that the virus used for the second immunity test was active, as the control guinea pigs developed typical symptoms and died on the fifth day.

This experiment indicated that large doses of unusually active virus given subcutaneously might induce the disease in some young horses. There appeared to be no distinct advantage in the simultaneous serum-virus method over the virus-alone method.

It was again evident that immunity is not sufficiently established until at least the 20th day after vaccination to withstand intracranial exposure to virus without symptoms.

Serum-Virus Neutralization

Howitt,[3] working with the "western" type of encephalomyelitis virus, reported that the serums of four horses convalescent from the western type of encephalomyelitis did not neutralize the western virus *in vitro* although the serums of two other horses possibly showed weak *in vitro* activity. Recently, Ten Broeck and Merrill[4] reported the *in vitro* neutralization of the "eastern" type of virus by the serums of horses which had recently recovered from the eastern type of encephalomyelitis. They reported also that the eastern and western strains of encephalomyelitis virus are immunologically dissimilar.

As we had a number of immunized horses which had survived either intracranial or intranasal virus exposure without symptoms, as well as five horses in the second experiment which had had a mild attack of the disease following the immunity test but eventually recovered, it was believed that they would afford ample material for study in connection with the serum-virus neutralization test.

Our test was similar to the serum-virus neutralization test of poliomyelitis given by Shaughnessy, Harmon and Gordon[5] and that reported by Howitt[3] for equine encephalomyelitis.

A stock virus suspension of 20 per cent horse brain in 50 per cent buffered glycerin (pH 7.5) was prepared, using virus B. A

standard mixture of 1 cc of a 1:1,000 final dilution of brain-tissue virus in phosphate saline and 1 cc of serum was finally adopted and used in all of the tests. The serum-virus mixtures were incubated two hours at 37° C. and then placed in the refrigerator at 5° C. for two hours. Guinea pigs then were injected intracranially with 0.2 cc of the serum-virus mixtures. Control guinea pigs were injected with the same dose of virus without serum.

The serum-virus neutralization test was used on the serums from 22 virus-vaccinated horses and 18 non-vaccinated controls, or a total of 40 horses. Virus neutralization occurred with the serums of only three horses: mare 8543, which had received 20 cc of the very active virus B; mare 8359, which had received virus A and B, and her colt which was born subsequent to immunization.

The serum of five other colts foaled by immune mares subsequent to immunization and one from a control mare failed to neutralize virus even while nursing the virus-resistant dams. One of the six colts (8550) died of encephalomyelitis twelve days after a single intranasal dose of 5 cc of a 1 per cent brain-tissue virus suspension while still nursing mare 8543, whose serum neutralized virus in a dilution of 1:5.

Even though the neutralization tests were repeated with the serum from the non-neutralizing immunized horses, using virus dilutions as high as 1:10,000, neutralization failed to occur.

These results indicate that even though horses may be resistant to infective doses of virus injected intracranially, neutralization of virus *in vitro* with their serum may not occur.

Resistance to virus infection and virus neutralization by the blood serum appear to be separate entities. This does not wholly apply to hyperimmune horses on serum production. It was found that 1 cc of a 1:20 dilution of two lots of encephalomyelitis serum produced at this station in 1932 would neutralize 1 cc of a 1:1,000 dilution of the stock virus.

Immunization of Ranch Horses

On the basis of the apparent safety and proven immunizing effect of subcutaneous live virus injection on highly susceptible horses as already set forth above, it was decided to try out the method in the field. An area was chosen where encephalomyelitis had been quite prevalent during 1931 and 1932. No effort was made to select the horses used on a basis of previous prevalence of the disease on the individual ranches, but the widest possible geographic distribution of ranches within the total area was aimed at. Nor was any attempt made to select the horses

on each ranch upon a basis of individual history as to encephalomyelitis. With a few exceptions, all horses on the ranch were injected either at the same time or by groups according to ranch operating conditions.

The animals used included those which had survived previous attacks of the disease, those previously more or less exposed to natural infection, young stock born since the last outbreak and some brought to the ranches from areas where the disease had not been active. All ages, from sucklings to aged animals of both sexes, including a few stallions, were represented.

The first lot of virus suspension was prepared from selected portions of two horse brains and two entire guinea pig brains, totaling 80 grams in weight. All four brains represented strain B virus, previously referred to as the most virulent we have encountered to date. This material was finely ground and made up to a 20 per cent suspension in Locke's solution, strained through gauze and stored at 5° C. until used. Every precaution was taken to avoid contamination during preparation. At the time of use this material was diluted further with sterile Locke's solution to a 2 per cent suspension and immediately injected subcutaneously. The material as finally injected therefore was a suspension of virus-bearing brain tissue entirely unmodified by heat, chemicals or other processing, except grinding and moderate dilution. It was free of bacteria on cultural test.

Beginning July 1, 1933, a total of 225 animals on different ranches received this material in doses of 5 cc for young colts and 10 cc for all others. All the injected animals were kept under fairly close observation for at least ten days, including in many cases the taking of at least one post-injection temperature.

With five exceptions, no very striking or definite reactions were noted though in some instances slight temperature rises and sluggishness were noted which rapidly disappeared. Five of the animals developed typical cases of encephalomyelitis of a particularly virulent and acute type within six to eleven days following the injection of the virus and all died, in spite of good clinical treatment, including antiserum, by experienced veterinarians.

One animal which died was a very young suckling colt at the institution ranch previously referred to whose dam was immunized in 1932. As the disease did not appear on the premises in 1933, the death of this colt was almost certainly the direct result of the 5-cc virus suspension injection.

Another fatality was a yearling whose dam had a severe attack of the disease during the time she was suckling him in 1932. As

there were no cases of the disease on this ranch in 1933, this death also was presumably the direct result of the 10-cc virus suspension injection. It is interesting to note that this animal acquired no visible evidence of infection or immunity while suckling the actively diseased dam during the previous year.

Another animal lost was a mature mare on a ranch where the disease had been prevalent since 1928, ten cases occurring in 1932. As a spontaneous case occurred in a non-vaccinated horse on the same premises twelve days later, the factor of natural infection as a complication in this instance cannot be excluded.

The two other animals dying were mature mares on a ranch where cases of encephalomyelitis had appeared periodically during 1931, 1932 and 1933, up to the time the animals were injected, so that in this instance also natural infection as a complicating factor cannot be excluded. None of the 225 animals injected developed encephalomyelitis from any source, natural or artificial, subsequent to the eleventh day.

A survey of the results with these 225 animals indicated that beyond reasonable doubt, two and possibly all of the five fatalities were the direct result of the method used. It also seemed probable that the death-rate of slightly over 2 per cent was due to the use of too virulent a strain of virus or too large a dose, or both. With the view of overcoming these factors, another lot of antigen was prepared.

For this purpose, tissue from a single horse brain carrying virus A, the less virulent strain of virus previously referred to, was selected. This material was prepared in the same manner and administered as a 2 per cent suspension of brain tissue. Starting late in July, 1933, 226 horses of the same age, sex and class, located in the same area as the previous group, were treated. The dose was as before, 5 cc for colts and 10 cc for older animals injected subcutaneously. No ill effects in the way of post-injection reactions were noted. An additional lot of 62 head also were treated with another lot of antigen prepared with the same strain of virus without ill effect. None of this total of 288 head developed encephalomyelitis from natural infection later.

Unfortunately from the experimental point of view, encephalomyelitis did not become prevalent in the area where these 513 injected horses were located during 1933, so there was no way of evaluating their resistance to natural infection. Judging from the almost solid immunity developed by the two controlled experimental groups, previously described, to the most severe type of experimental infection, they would have been fully protected.

DISCUSSION

An analysis of the results obtained with a grand total of 591 equines under varied laboratory and field conditions seems to demonstrate that the subcutaneous injection of the unattenuated virus in the form of dilute brain suspension confers an active and practically solid immunity against the western type of encephalomyelitis. The duration of this immunity has not been determined definitely but it appears adequate to protect against the severest exposure to natural infection for a period fully as long as that involved in any seasonal epizoötic reported to date. If proper precautions are taken, the loss from the disease as a result of such injections should be practically nil. No other method of conferring a satisfactory immunity has so far been reported.

In attempting the immunization of equines by the method we have outlined, certain guiding principles should be followed closely. The strain of virus used should be one whose behavior is well standardized by an adequate number of horse and guinea pig passages in the laboratory; also it should have proved non-invasive when injected by the subcutaneous route. Observations in connection with three low-virulence strains have shown that they are fully as antigentic as the highly virulent or invasive ones and certainly safer for field use, exalted virulence having apparently no advantage from the antigenic standpoint.

Dosage should be reasonably conservative. There seems to be no very direct relationship between the amount of virus-bearing brain tissue injected at one time and the immunity produced. Given a satisfactorily active virus, a dosage of 5 cc of a 2 per cent suspension of brain tissue is probably adequate, this amount to be reduced to 2 or 3 cc for colts under six months of age. Expressed in another way, a dosage of 1,000 to 2,000 guinea pig m. l. d., as determined by intracranial injection on these animals, appears to confer a satisfactory immunity in equines, although in a majority of cases we used a larger dosage.

We have already reported a routine method of propagating and storing equine encephalomyelitis virus.[1] Immunization studies necessitated further work in connection with the viability of this virus under various conditions.

Virus strains A and B previously mentioned in this paper were used in this work which may be briefly summarized as follows:

A 20 per cent suspension of finely ground horse brain tissue virus in 50 per cent phosphate glycerin (pH 7.5) will remain active for at least four months when stored at 5° C. The same ma-

terial remained active for only 14 days at 25° C. Similar virus suspensions containing 0.5 per cent phenol remained active for three months when stored at 5° C.

The 20 per cent ground brain virus in 50 per cent phosphate glycerin constitutes a very satisfactory stock material which may be kept in cold storage or taken to the field in properly iced containers and there diluted with phosphate salt solution* to the desired concentration just prior to injection. It should be emphasized that this virus is rapidly inactivated by slight acidity and a pH of 7.3 to 7.5 should be maintained in all solutions used for storage, emulsification and dilution.

Finally we wish to stress again certain points for the consideration of anyone inclined to try the method of immunization we have described. The nature of the virus used requires adequate laboratory facilities and makes a closely coördinated laboratory and field personnel essential. While we believe the risk of artificial infection to be negligible if all proper precautions are observed, this factor should always be considered.

It would appear that this method of immunization is in many instances preferable to attempts at control by the prophylactic use of antiserum, with the very brief and uncertain passive immunity it confers, in case of an actual or threatened severe outbreak of encephalomyelitis. Final decision as to the use of the method described must be based on a careful consideration and evaluation of all the factors involved in each instance.

As to whether the results we have obtained are applicable to the eastern strains of encephalomyelitis virus and the disease it causes in the case of horses we have no basis for an opinion. Very limited experiments with guinea pigs showed that the subcutaneous injection of eastern type virus strains produced no reaction but was followed by a solid immunity to intracranial injection with the same virus.

Conclusions

1. A method of actively immunizing equines against the "western" type of equine encephalomyelitis has been presented.

2. The immunity produced within 21 days appears more than ample to protect against natural infection for a period longer than the prevalence of the disease during any one season in a given locality.

*During the earlier part of this work, Locke's solution was used as a diluent for stock virus. Subsequent observations showed that phosphate salt solution, pH 7.3—7.5, was more stable under sterilization and storage and its use is recommended.

3. Spontaneous virus infections did not occur among control horses remaining in contact with virus-injected horses during a period of seven months.

4. Resistance to natural or artificial infection and the power of the serum of an animal to neutralize virus *in vitro* do not appear to be co-related.

5. The method as so far developed is not suitable for general unrestricted or commercial use.

6. Whether or not the method outlined can be applied to the "eastern" type of encephalomyelitis and the disease it produces in horses has not been determined.

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Index Veterinarius

The first issue of this new quarterly index to veterinary publications reveals that there are more than 1,000 magazines, bulletins, pamphlets and reports in circulation, dealing with subjects of interest to veterinarians. Volume I, No. 1, of the *Index Veterinarius*, although dated April, 1933, and covering the first three months of that year, was distributed only recently. It is edited by W. A. Pool, M. R. C. V. S., and published by the Imperial Bureau of Animal Health, Weybridge, Surrey, England. The material, which is complete in every detail, includes not only names of publications, but names of authors and titles of articles. References are fully cross-indexed. The book, covering 304 mimeographed pages, is easy to read, because of a carefully planned layout. It is attractively bound, and priced at £4 for an annual subscription. It should be worth the price to anyone who desires to keep abreast of current veterinary literature.

Mood-y

Teacher: Johnny, take this sentence: "I led the cow from the pasture." What mood?

Johnny: The cow, ma'am.

SORE MOUTH IN SHEEP TRANSMISSIBLE TO MAN*

By I. E. NEWSOM *and* FLOYD CROSS
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Schmidt and Hardy, in their bulletin on sore mouth in sheep,[1] quote Willems, of Belgium, as suggesting the possibility of transmission of sore mouth to man. They refer also to an article by Hatziolos, of Greece, in which he reported the transmission of the disease to shepherds with the "occurrence of stomatitis, with vesicles on the gums, tongue, cheeks and lips." Schmidt and Hardy say: "We have observed two similar instances but as yet have no definite proof that such lesions were due to sore-mouth virus affecting sheep or goats."

Brandenberg,[2] in 1932, describes an outbreak of lip-and-leg ulceration in sheep in North Dakota, in which the disease apparently was transmitted to two men who were engaged in treating the flock. The lesions developed "on the hands and arms to the elbows and on the legs to the knees. Large lentil-like nodules appeared, which were slightly painful. There was a deep smarting or itching sensation if they were pressed upon or irritated in any way. There was swelling of the neighboring lymph-glands." As one of these men was a student at the State College, some bacteriological work was done on his lesions in that institution but without definite results.

The cases which we wish to present occurred in the vicinity of Fort Collins, in November, 1933. A sheep feeder and his two Mexican helpers treated a large band of sheep for sore mouth, it taking some three days to finish the job. It was noticed that the sheep carried a great many cockleburs in the wool, and, as a result, the hands of the workers were almost continually irritated. Four days after the treatment was ended, or on November 22, several large vesicles appeared on the hands of the feeder. He

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consulted a physician who sent him to our laboratory. At this time nine fairly well developed vesicles had appeared on the right hand, with a smaller number on the left. The larger vesicles were composed of several compartments and were surrounded with a reddened zone and moderate swelling. Complaint was made that there was some swelling of the lymph-glands in the armpits, with considerable pain in that region. The vesicle on the index finger just back of the nail, which appears black in figure 1, seemed to be most painful, possibly because the skin was very much thickened at that point and the vesicle was more compressed. The larger lesion was opened at several points and emitted a thin, limpid fluid. Three swabs were soaked in the

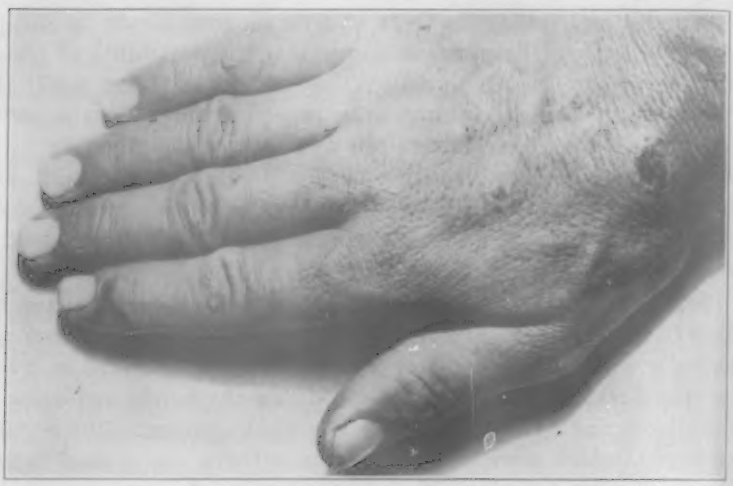

FIG. 1. Hand of man who developed vesicles after treating sheep with sore mouth. Taken 12 days after vesicles appeared.

fluid, put back into their respective containers and kept for further work. The accompanying picture was not taken at that time, but twelve days later, after the lesions had dried up to a considerable extent. He stated at this time that the helpers were showing similar symptoms but not so severe as his own.

On the following day, a scratch was made on the inside of the thigh of a lamb in our experiment pens and one of the swabs was rubbed into the scratch. Five days later, there was considerable reddening at one end of the scratch. By the next day, pustules had appeared along the entire tract. These had attained the maximum development eleven days after inoculation, at which time the accompanying photograph (fig. 2) was taken. Fol-

lowing this, a rather large scab developed, which fell off in due time.

Twelve days after the swabs were taken, another sheep was inoculated in a manner similar to the first. Four days after the inoculation, two vesicles had developed on the scratch. These continued through the usual course of pustule and scab formation but showing much less severity than the previous inoculation.

FIG. 2. Row of pustules on thigh of sheep taken 11 days after inoculation from hand of man.

DISCUSSION

Since sore mouth is one of the most common diseases of feeder lambs and is consequently very widespread, and since it is not infrequently treated by feeders, it would seem that it is not easily transmitted to man, else previous reports would have come to us. There seems no doubt in this instance, however, that the disease was actually transmitted to this feeder and his helpers, since in his case at least swabs taken from the vesicles reproduced the disease in two different sheep. Probably the cockleburs in the wool had something to do with the readiness with which the men were inoculated. This feeder has handled lambs for some 15 years previously, without suffering any such symptoms.

Only palliative treatment was used by the physician in charge, followed by an uneventful recovery after about three weeks.

REFERENCES

[1]Schmidt, H., and Hardy, W. T.: Sore mouth (contagious ecthyma) in sheep and goats. Tex. Agr. Exp. Sta. Bul. 457 (1932).

[2]Brandenburg, T. O.: Lip-and-leg ulceration in sheep with report of two cases in man. Jour. A. V. M. A., lxxxi (1932), n. s. 34 (6), pp. 818-820.

ERADICATION OF BANG'S DISEASE IN A HERD OF BEEF CATTLE*

By C. C. PALMER, *Newark, Del.*

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As the practicability of blood testing and removal of the reacting animals from the herd as a means of eradicating Bang's disease continues to be a subject of considerable controversy, and owing to the fact that the collection of blood samples from beef cattle presents problems differing somewhat from those encountered in dairy cattle, this report upon the eradication of the disease in a herd of beef cattle is submitted.

In 1930, 33 Hereford cows and three bulls constituted the breeding herd. The growing herd, consisting of approximately 70 calves, yearlings and two-year-olds, was maintained in pastures and stables well isolated from the breeding herd. The cows were bred to calve during the spring months. During the winter of 1929-30, many of the cows of the breeding herd aborted. On May 6, 1930, blood samples were collected from the breeding herd. The agglutination test for Bang's disease revealed that 23 of the cows and one bull were positive to the test, three cows were suspicious, and seven cows and two bulls were negative to the test. The positive and suspicious animals were sold for slaughter and the negative animals were placed in new quarters which were isolated from those occupied by the growing herd. The old lots occupied by the breeding herd remained vacant for a few months.

Twenty-one days after the original test, blood samples were collected from the seven cows and two bulls which were negative on the first test. These animals were again negative. On the same date, blood samples were obtained from 18 heifers of the young herd. These animals had been selected to replace the cows in the breeding herd which had reacted at the first test. However, they were not removed from the growing herd. They were negative to the agglutination test.

The breeding herd, now consisting of 25 females, was tested again after an interval of 18 days. The 18 new additions to the breeding herd were again negative to the test. One of seven

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original herd cows held in isolation gave a suspicious reaction to the test. This animal was sold for slaughter.

In December, the annual tuberculin test of the herd was due. On December 1, while making this test, blood samples were collected from all of the animals in the herd. Eighty-one animals were tested. Every animal in the herd passed a satisfactory tuberculin test and all animals, except two, passed satisfactory agglutination tests for Bang's disease. The two positive animals were a cow belonging to the original negative animals of the breeding herd, and a bred heifer, a member of the new breeding herd. The new breeding herd, consisting of 18 two-year-old heifers, had been transferred two months previously to the quarters formerly occupied by the original breeding herd. The two reactors were sold for slaughter.

At the next test, made on April 17, 1931, all animals except newborn calves, which were not tested, passed satisfactory tests for Bang's disease. On this date, seventy-nine animals were tested. Since the above test, the herd has been tested semi-annually and no reactors have been found. The last test, conducted upon blood samples collected on December 18, 1933, revealed that the herd is comprised of 120 negative animals. As the last reactors were found in the herd on December 1, 1930, the herd has been negative to the agglutination test for Bang's disease for three years.

Method of Collecting Blood Samples

The collecting of blood samples from beef cattle presents problems in restraint differing somewhat from those usually encountered in dairy herds. To aid in this work, a special made crate was employed. The cattle to be tested were driven into a small enclosure, as a stable or pen, and by means of panels were driven into the crate one at a time. As soon as the animal was completely within the crate, bars were inserted behind the animal to prevent it from backing out of the crate. The head of the animal was secured by means of a tong bull-leader, pulled through the opening at the front of the crate, then to one side where the rope attached to the leader was made fast. This position facilitated the insertion of a hypodermic needle into the jugular vein. After collecting the blood sample, the head of the animal was freed and the gate at the front of the crate was opened, permitting the animal to pass out of the crate.

A crate to be used for this work should be made of strong pieces of lumber held together by bolts. The size of the opening at the front end of the crate above the gate should be adjustable.

If this opening is too large, young cattle will climb through the space and, once they have started, it is extremely difficult to force them back into the crate. The opening should have a height sufficient for passage of the head and neck but no higher. The gate at the front of the crate should be high enough to prevent escape of the animal and yet low enough not to interfere with easy access to the jugular vein. The crate should have a heavy plank floor. Although such a crate is very heavy, it can be handled by several men and thus moved from place to place. The crate shown in figure 1 has been in use several years. It was built specifically for the purpose of holding beef cattle. It is used in dehorning, tuberculin-testing and blood-testing.

FIG. 1. Crate for restraining beef cattle.

DISCUSSION

This herd was not tested for Bang's disease prior to the severe wave of abortion that occurred during the winter and spring months of 1930. It is believed, however, that the infection was not present prior to that time as there is no history of abortions occurring within the herd up to that time. The infection was introduced probably by two dairy cows which were added to the herd as nurse cows in 1929. When the Hereford cows started

aborting, the dairy cows were suspected of being responsible for the infection and accordingly were removed from the herd. Thus, an opportunity was not afforded for ascertaining the soundness of this theory.

Starting with a herd in which 75 per cent of the breeding animals were infected with Bang's disease, it was not a difficult problem to eradicate the disease from the herd. No attempts were made to destroy the infection in the stables, yards or pastures used by the infected animals other than discontinuing their use for cattle for a few months. This period of time evidently was not long enough, as one heifer became infected when placed in the quarters formerly occupied by the infected herd.

A properly constructed crate, into which each animal to be tested is driven, greatly facilitates collection of the blood samples. As such a crate has proven very practical for tuberculin-testing, it was found to be an advantage to run the Bang's-disease and tuberculin tests simultaneously.

Conclusions

1. Bang's disease was controlled and eradicated by agglutination-testing and removal of the positive animals in a herd of Hereford cattle in which 75 per cent of the breeding stock was found infected.
2. Blood-testing and removal of the reactors proved entirely satisfactory and practical in the eradication of Bang's disease in this herd.
3. The herd has remained free of all evidence of Bang's disease for a period of three years.
4. The use of a properly constructed crate facilitates the collection of blood samples in beef cattle.

MARKED TENIASIS IN A DOG*

By L. B. Sholl, *East Lansing, Mich.*
Michigan State College

The subject is an eight-month-old female Beagle hound. The animal was used a great deal for rabbit hunting during the past winter. According to the owner, the dog apparently was in good health until two days ago. The appetite failed suddenly, no treatment was given, and the dog died on the second day of illness. The owner was certain that someone had given the dog poison or ground glass.

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Autopsy: The animal is in fair flesh. The skin and subcutaneous tissues are negative. The mesenteric lymph-nodes are congested. All other lymph-nodes are negative. The head, neck and chest are negative. The peritoneum over the stomach and intestines shows marked congestion. The spleen and pancreas are negative. The liver shows some congestion and fatty changes. The stomach is almost empty. Some reddish mucus is present, and the mucosa is congested. Some hemorrhages are noted in some of the folds. In the small intestine there are 107 tapeworms (*Taenia pisiformis*), and a marked hemorrhagic inflammation extends throughout the intestine. The kidneys are congested and show some evidence of cloudy swelling. The urinary bladder and genital organs are negative.

This case is an interesting one due to the suspicion of the owner, the apparently short duration of illness, and the extremely heavy infection with tapeworms.

BILATERAL LUXATION OF THE FEMURS IN A CALF*

By L. B. SHOLL, *East Lansing, Mich.*
Michigan State College

The subject is a four-month-old female Jersey calf. Parturition occurred at pasture. The calf was apparently normal, and was allowed to remain with the cow during the first week of life. Then she was put in a horse-stall in the barn and has been there since, with little or no exercise and no sunlight. After being put in the barn she was weaned gradually, and the owner has fed her liberally on skim milk, alfalfa, corn and oats. About 18 days prior to examination, the owner found the calf down and unable to rise. The owner knew of no possible source of injury. She gradually became able to stand but has not been able to advance the right rear leg, which lies across the left hock.

Examination: The calf is in fair flesh and appears normal except for marked deformity of the hind quarters. The right tuber coxæ is considerably lower than the left. The right leg is held backward and across the left leg, and the calf is unable to move it past the left leg. On manipulation of the hind parts there is a marked crepitation on both sides, suggesting fracture of the pelvis or femurs. The owner is advised to allow slaughter.

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Autopsy: No gross lesions are noted in any of the internal organs. The ribs and long bones show no gross evidence of rickets. The pelvis is markedly deformed. The symphysis of the ileum, pubis and ischium is much enlarged, projecting into the pelvic cavity quite markedly. The right pubis is much shorter than the left, and the right acetabulum is markedly deformed. A considerable amount of fibrin and some clotted blood are present in the joint cavity, which is quite largely filled in with excess connective tissue and cartilage. The head of the right femur is luxated upward, its cartilage is markedly eroded, and it rests against an irregular, bare, bony area above the acetabulum. The round ligament is destroyed. The left joint is similarly but more mildly involved.

FIG. 1. Calf with luxation of both femurs.

Bulletin on Fowl Typhoid

An article on "La Typhose aviaire à Stanleyville," by Dr. G. C. Bourguignon, director of the Laboratory of Bacteriology and Parasitology, at Elisabethville, Belgian Congo, which appeared in the *Bulletin Agricole du Congo Belge* for June, 1933, may be of interest to veterinarians in the United States. Those interested in looking over this article may obtain copies of the *Bulletin* by writing to the Ministère de l'Agriculture, Place Royale, Brussels, Belgium.

12th International Veterinary Congress
New York—August 13-18, 1934

PATHOLOGISCHE MIKROSKOPIE. (Microscopic Pathology.) Oskar Seifried, Professor, des Institutes für Tierpathologie der Universität München, und Ed. Heidegger, Assistant, des Institutes für Tierpathologie der Universität München. 78 pages. Ferdinand Enke Verlag Stuttgart-W, 1933. Unbound, 3.9 RM.

This small book by Drs. Seifried and Heidegger aims to give the essential processes in the preparation of tissues for microscopic examination. The authors state in their preface that only such methods are given as have been verified in the dissecting-room or laboratory. A short account of bacteriological methods is included. All methods given are very briefly described. It is difficult for one to understand exactly the group of individuals that should use this book. It is too brief for one engaged in laboratory investigation in the field of microscopic pathology, and it is not in our judgment sufficiently detailed for the practitioner of veterinary medicine in the field. It is intended in accordance with the authors' preface for students and "beginners" in this field. The material is arranged in an orderly manner and the methods are briefly and concisely told. It is in no sense a reference work such as are Mallory and Wright's Microscopical Technique and Microscopical Technique, edited by McClung.

C. P. F.

BRUCELLA INFECTIONS IN ANIMALS AND MAN. Methods of Laboratory Diagnosis. I. Forest Huddelson, Department of Bacteriology and Hygiene, Michigan State College. xiv + 108 pages, with 24 figures (2 in color). The Commonwealth Fund, New York, 1934. Price, $2.25.

Considering the attention which has been focused upon the Brucella infections recently, this monograph by an outstanding authority on the subject is very timely and should receive a warm welcome from veterinarians, physicians and laboratory workers.

An immense amount of useful information has been crowded into this small handbook. In this connection, the author points out that the omission of certain materials pertaining to the subject was done, not with the idea of casting any reflection on its

value, but with the thought that the inclusion of too much material would merely lead to confusion and detract from the purpose for which the manual was intended.

In order to give the best idea of the ground covered in this book, it will suffice to mention the titles of the seven chapters: The Genus Brucella; Methods of Isolating Brucella; The Pathology of Brucella Infections; Serological Methods of Determining Brucella Infections; Allergic Methods of Determining Brucella Infections; Method of Determining Brucella Infections by Measuring the Opsono-Cytophagic Power of the Blood; Methods of Differentiating the Species of the Genus Brucella. In addition, there is appended a bibliography of 189 titles.

The book is printed on an excellent stock of paper, the typography is pleasing and the illustrations are well selected and splendidly reproduced. To anyone who has occasion to refer to the latest information on the Brucelloses, this monograph commends itself.

BEATING WINTER. J. F. DeVine. 336 pages, 66 figures and 7 maps. Goshen Democrat Printing Co., Goshen, N. Y., 1934. Price, $2.50 plus postage.

A travelogue in which are recounted the rovings of a party who participated in a Mediterranean cruise in 1932. Historical data are interspersed in a pleasing manner with the log of the tour, which included most of the countries in southern Europe, northern Africa and western Asia. Many interesting incidents are related in the charming style of the veterinarian-author who is known to practically every member of the profession in America.

As an appendix to the book, there is an account of the European tour taken in 1930 by the party of American veterinarians and their wives previous to attending the Eleventh International Veterinary Congress in London.

Rooster Without Wings

A rooster, born without wings and now grown to adulthood, is the gift of Mrs. Olia Deering, of Rose Hill, Ky., to the Smithsonian Institution. It has been given quarters at the National Zoölogical Park where it is being studied by Dr. Herbert Friedmann, curator of birds at the U. S. National Museum, according to *Science*. Fowls without wings are hatched occasionally, but all hitherto reported have died while still young chicks. The present specimen, a Plymouth Rock, is a healthy bird. Its parents were normal.

OBSERVATIONS ON THE FIRST STAGE LARVAE OF GASTEROPHILUS INTESTINALIS IN TONGUES OF HORSES IN CENTRAL IOWA. E. F. Knipling. Jour. Parasitol., xx (1934), p. 196.

Postmortem examination of the tongues of horses at various intervals was made to determine the extent of larval infestations of *Gasterophilus intestinalis*. This study was made to determine how long, after oviposition has ceased, the stomach of the horse may be reinfested from viable larvae in the eggs or from larvae already in the mucous membrane of the tongue. After December 15, there was a rapid decline in the average number of larvae found. Since very few larvae were found after December 15, tongues examined 30 days later showed the presence of few larvae. The author suggests that it is essential to destroy eggs on horses if they are treated with carbon disulfid before January 15, as observations indicate that the number of viable eggs on the host up to December 15 is sufficient to produce a reinfestation.

VIRULENCE OF SALMONELLA PULLORUM. Wayne N. Plastridge and Leo F. Rettger. Jour. Inf. Dis., liv (1934), 1, p. 23.

Marked differences were observed in the virulence of individual strains of *S. pullorum* for chicks, adult birds, rabbits and guinea pigs. Some strains were found to be highly virulent for both chicks and adult birds; others possessed a low degree of pathogenicity for chicks but were highly virulent for adult birds, and still others were relatively avirulent for both young and old chickens. Pronounced changes can be induced in the virulence of some strains by the action of bacteriophage. One particular strain which was moderately virulent for chicks and only slightly virulent for adult birds was rendered practically avirulent for chicks and highly virulent for adult birds by treatment with bacteriophage. This condition appeared to be dependent on the establishment of a certain balance between the activity of the bacteriophage and the growth of cells. In general, passage through animals had no appreciable effect on the morphologic, colonial and agglutinative characteristics of the variants em-

ployed. The feces of adult birds artificially infected with *S. pullorum* were found to contain the pullorum organism at irregular intervals when examined from 102 to 194 days following exposure.

ISOLATION OF BACTERIA OF THE BRUCELLA GROUP FROM APPARENTLY HEALTHY SWINE. William H. Feldman and Carl Olson, Jr. Jour. Inf. Dis., liv (1934), 1, p. 45.

Blood was obtained before slaughter from a group of 102 head of swine ranging in age from seven to twelve months. Serum from this blood was used to conduct agglutination tests, using the antigen of *Br. abortus*. The blood of two of the animals showed definitely positive reactions and these animals were subjected to careful postmortem examination. By the inoculation of guinea pigs an organism of the Brucella group was obtained from each of the animals which had reacted positively to the agglutination test. The organism was obtained from the lymph-nodes of the head and anterior cervical region and from an abscess of the spermatic cord of one of the animals. In the other animal the infective organism was secured from the spleen. Neither the lymph-nodes nor the spleen revealed morbid changes. The authors conclude that bacteria of the Brucella group may exist in the tissues of apparently normal swine without giving rise to discernible symptoms of disease.

EPIZOÖTIC FOX ENCEPHALITIS. V. General and pathogenic properties of the virus. R. G. Green, N. R. Ziegler, W. E. Carlson, J. E. Shillinger, S. H. Tyler and E. T. Dewey. Amer. Jour. Hyg., xix (1934), 2, p. 343.

The virus of fox encephalitis remains viable in an animal carcass for several days after death and may be stored in 50 per cent glycerin for several years without loss of virulence. The virus is easily filtrable through Berkefeld-N filters, and the specific inclusions typical of this disease occur in animals infected with filtrates. The susceptibility of the coyote to this virus appears equal to that of the red fox. The inclusion bodies found in the coyote differ considerably from those found in the fox. The gray fox is resistant to this virus as is the mink. Attempts to induce an infection with the virus in rabbits, even by special procedures designed to allow adaptation, met with failure. The virus appears nonpathogenic for white rats, squirrels, guinea pigs, and cats. Dogs have been found fairly susceptible to the virus.

Susceptibility and resistance in this animal often appear as litter characteristics. Intranuclear inclusions typical of this virus are produced abundantly in the dog, not only in the central nervous system but also in the thoracic and abdominal organs. Ferrets are resistant to infection with the virus. Sheep appear entirely immune. The virus seems to possess no invasive powers for the monkey.

STUDIES ON THE LEUCOCYTE CONTENT OF MILK DRAWN FROM BRUCELLA ABORTUS-INFECTED UDDERS. C. C. Prouty. Jour. Bact., xxvii (1934), 3, p. 293.

A study was made of the leucocyte content of the milk from 18 abortus-infected cows in relation to the presence of *Br. abortus* within the udder. Thirteen cows were found by cultural methods to harbor *Br. abortus* in one or more quarters of the udder. Two were found to be infected in all quarters and seven in only one quarter. Twenty-one of the 72 quarters were found to be infected. The average leucocyte count per cubic centimeter of milk from the 21 infected udders was 355,000, as compared to 343,000 for milk from the 51 quarters that gave negative cultural findings. Two of the *Br. abortus*-infected quarters and two of the non-infected quarters produced milk of a high leucocyte count, due to the presence of active streptococcic mastitis. When the samples of milk coming from these quarters were omitted in computing averages, average cell counts of 145,000 and 185,000 per cubic centimeter were obtained for the samples from the *Br. abortus*-infected and non-infected quarters respectively. Similar average leucocyte counts were obtained for samples of milk from animals in an abortion-free herd. All samples of milk giving negative agglutination reactions, in amounts of milk serum less than 0.08 cc, gave negative cultural findings for *Br. abortus*.

THE DURATION OF MALARIAL INFECTION IN BIRDS. Reginald D. Manwell. Amer. Jour. Hyg., xix (1934), 2, p. 532.

Observation of 118 cases of avian malaria, due to infection with five species of plasmodium (*cathemerium, praecox, circumflexum, elongatum* and *rouxi*), showed that the parasites persisted in the body of the host throughout the period of observation, which was not less than a year in any case, and in a few instances covered three years. The results indicate that great caution must be used in regarding any case of avian malaria as entirely recovered, since the parasite level during the chronic stage may be too low for microscopic detection, and occasionally even too low

for successful subinoculation unless relatively large amounts of the blood are employed.

SALMONELLA AERTRYCKE IN COLITIS OF FOALS. Philip R. Edwards. Jour. Inf. Dis., liv (1934), 1, p. 85.

An outbreak of infectious colitis among suckling foals was found to be due to infection by *Salmonella aertrycke*. This organism was isolated in all the fatal cases and from certain infected animals which recovered. The disease was characterized by a high temperature, marked depression, abdominal pain, and a fetid diarrhea. Blood appeared in the feces of some animals. Intense colitis was the most marked pathologic change. The large intestines were thickened and inflamed, and there was an almost complete necrosis of the mucous membrane. The colic lymph-glands were enlarged and congested. A vaccine prepared from cultures of the organism apparently had some value in controlling the infection.

EPIZOÖTIC FOX ENCEPHALITIS. VI. A description of the experimental infection in dogs. R. G. Green and J. E. Shillinger. Amer. Jour. Hyg., xix (1934), 2 p. 362.

Dogs may be experimentally infected with the virus of fox encephalitis. Irregularities have been encountered in attempts to transmit the disease that appears best explained on the basis of familial immunity. The fatal infection usually runs a short, violent course of less than a week in a manner similar to that of the infection in foxes. The lachrymal and nasal secretions of the dog tend to become purulent. The nervous symptoms in dogs are largely concerned with a state of excitement, often describable as a "running fit." The general pathology of the disease consists of a cellular infiltration in the central nervous system and focal necrosis of the liver. Specific intranuclear inclusions are found in cells of the vascular endothelium, meningeal cells, reticulo-endothelium, hepatic cells, and occasionally in the cortical cells of the adrenal.

THE ISOLATION FROM THE ROCKY MOUNTAIN WOOD TICK (DERMACENTOR ANDERSONI) OF STRAINS OF BACT. TULARENSE OF LOW VIRULENCE FOR GUINEA PIGS AND DOMESTIC RABBITS. Gordon E. Davis, Cornelius B. Philip and R. R. Parker. Amer. Jour. Hyg., xix (1934), 2, p. 447.

Three strains of *Bact. tularense* of low virulence for rabbits and guinea pigs were isolated from *Dermacentor andersoni*. Tests

show that these three strains are less virulent than two strains isolated from the rabbit tick, *Haemaphysalis leporis-palustris*. The results were the same whether judged by the data of the cutaneous vaccination of domestic rabbits with pure cultures, the cutaneous vaccination of guinea pigs with pure cultures, or the cutaneous vaccination of guinea pigs with spleen pulp. None of the rabbits died following injection of the *D. andersoni* strains, whereas all died when injected with the *H. leporis-palustris* strains. All five strains killed all guinea pigs inoculated, but the survival was longer when the *D. andersoni* strains were used.

THE BLOOD FORMATION AND THE COPPER CONTENT OF THE CHICKEN EMBRYO. S. Sumegi. Abst. Arch. Path., xvii (1934), 2, p. 246.

The copper content of chick embryos during incubation increases gradually up to the time of the onset of respiration. Beginning from the onset of respiration, the copper content increases more rapidly. The number of red blood cells and their hemoglobin content are in direct proportion to the copper content of the embryo. The morphologic maturity of the blood cells is almost in direct proportion to the copper content. Twenty-four hours after the chicken is hatched, the copper reserve is markedly diminished.

THE DOG AND THE "BUTTON FEVER" VIRUS. Paul Durand. Abst. Arch. Path., xvii (1934), 2, p. 263.

The dog was studied as a potential reservoir for this typhus-like disease, apparently transmitted to man by the dog tick. In some instances experimental infection was induced, with a positive Weil-Felix reaction. Dogs showed little or no reaction, but successful transfer to man (fever therapy in dementia praecox) was accomplished by subcutaneous injection of dog's blood.

THE FILTERING CAPACITY OF LYMPH-NODES. Cecil K. Drinker, Madeleine E. Field and Hugh K. Ward. Jour. Exp. Med., lix (1934), 4, p. 393.

In anesthetized dogs the popliteal lymph-node alone, and the popliteal and iliac lymph-nodes in series, were perfused with solutions containing erythrocytes and streptococci. The perfusions were carried out under conditions of lymph-flow and pressure within the limits of those occurring in the actively moving dog, or after a severe degree of inflammatory swelling developed.

The experiments indicate that normal lymph-nodes possess a high degree of filtering efficiency, so great as to make it fairly certain that, in a part kept at rest early in an infection, practically no microörganisms would escape the nodes in the line of drainage.

LYMPHOMATOSIS, MYELOMATOSIS AND ENDOTHELIOMA OF CHICKENS CAUSED BY A FILTRABLE AGENT. II. Morphological characteristics of the endotheliomata caused by this agent. J. Furth. Jour. Exp. Med., lix (1934), 4, p. 501.

When stimulated by a filtrable agent of leukosis of chickens (strain 2), endothelium may undergo seemingly unrestricted growth. These neoplasms of endothelium are usually unaccompanied by the formation of blood cells. Occasionally they produce hemocytoblasts, discharged like those of the normal marrow into vascular channels as also myelocytes about the vessels. The same agent that stimulates endothelium also stimulates erythroblasts, myelocytes and hemocytoblasts to unrestricted growth without obviously affecting the endothelium; and the association of endothelioma and leukosis is the result of stimulation of several types of cells by a single virus. Myelocytes appear also to develop from mesenchymal or endothelial cells without the intermediary stage of hemocytoblasts. It is often impossible to determine whether the neoplasms caused by the virus of strain 2 are of endothelial or mesenchymal origin, and it is possible that both types of cells may be stimulated by the same virus. Types of sarcoma like those described by Rous are not produced by the virus of strain 2.

On Binoculars

A compact and valuable booklet on binoculars has been issued recently by the Bausch & Lomb Optical Co., of Rochester, N. Y. This booklet is both a catalog and compendium of information on binoculars. Progress in the construction of binoculars is traced through the years; steps in the making of the present high-quality instruments are described and illustrated with photographs; suggestions on choosing the right kind of model are made; and, of special interest, a glossary of optical terms is included.

Those interested in owning a copy of the booklet should write to Bausch & Lomb. The title is "Life Long Binoculars."

12th International Veterinary Congress
New York—August 13-18, 1934

Regular Army

First Lt. Austin T. Getz is relieved from further assignment and duty at Fort Des Moines, Iowa, and will proceed, on or about April 1, 1934, to Langley Field, Va., for duty.

Orders assigning Colonel Wm. A. Sproule to Fort Geo. G. Meade, Md., upon his arrival in New York City, are amended so as to direct him to report to the commanding general, New York port of embarkation, Brooklyn, N. Y., for duty.

Captain Laurence R. Bower is relieved from further assignment and duty at the New York port of embarkation, Brooklyn, N. Y., effective on or about April 1, 1934, will then proceed to Fort Geo. G. Meade, Md., and report to the commanding officer, for duty.

Major Allen C. Wight is relieved from duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about July 5, 1934, will then proceed to Carlisle Barracks, Pa., for duty.

Major Robert P. McComb, Fort Logan, Colo., has been directed to report to the president of an Army retiring board at Fitzsimons General Hospital, for examination by the board at such time as the president thereof may designate.

The promotion of Lt. Col. James R. Shand to the grade of colonel with rank from February 11, 1934, is announced.

Each of the following named second lieutenants of the Veterinary Corps is relieved from further assignment and duty as student, Medical Field Service School, Carlisle Barracks, Pa., effective upon completion of his present course of instruction, on or about June 5, 1934, and will proceed to the station indicated after his name and report not later than June 30, 1934, for duty.

Robert A. Boyce, Jr., Front Royal Quartermaster Depot, Front Royal, Va.

Clarence L. Taylor, Fort Hoyle, Md.

Second Lt. Wayne O. Kester is relieved from further assignment and duty as student, Medical Field Service School, Carlisle Barracks, Pa., effective upon completion of his present course of instruction, on or about June 5, 1934, and will proceed to Washington, D. C., and report not later than June 30, 1934, to the commanding officer General Dispensary, United States Army, for duty, and in addition will act as attending veterinarian at the Army War College and Bolling Field, D. C.

Lt. Colonel Horace S. Eakins is assigned to duty at Fort Clark, Texas, effective upon completion of his tour of foreign service in Panama.

Major James R. Sperry is relieved from further duty at Reno quartermaster depot, Fort Reno, Okla., and directed to proceed to Madison Barracks, N. Y., and report for duty not later than May 31, 1934.

Major Floyd C. Sager is relieved from further assignment and duty at Madison Barracks, N. Y., effective in time for him to proceed to Lexington, Ky., and report for duty with the purchasing and breeding headquarters not later than May 31, 1934.

Veterinary Reserve Corps

New Acceptance

Sisk, David Ephram.....Capt.....Mansfield, Ill.

Promotion

To

Hudson, Bentley Farnell....Capt.....105 N. Main St., Moweaqua, Ill.

Transferred from FA Reserve

Thames, Curtis Bush.....1st Lt.....Box 212, Greenville, Ala.

BUREAU TRANSFERS

DR. W. C. NYE (Colo. '20) from Boise, Idaho, to Havre, Mont., on field inspection.

DR. J. E. GRIFFIN (McK. '18) from Saint Joseph, Mo., to Saint Paul, Minn., on tuberculosis eradication.

DR. L. BILIKAM (San Fran. '17) from Belmae Park, Calif., to Honolulu, T. H., on meat inspection.

DR. SALMAR P. BOLSTAD (K. C. V. C. '12) from Milwaukee, Wis., to Valentine, Neb., on meat inspection.

DR. MIHALY BORSOS (San Fran. '14) from Chicago, Ill., to Jersey City, N. J., on meat inspection.

DR. CHARLES WEBSTER (Colo. '14) from Scott City, Kan., to Hugo, Colo., on field inspection.

DR. C. T. ADAMSON (Iowa '29) from Lincoln, Neb., to Saint Paul, Minn., on tuberculosis eradication.

DR. A. L. HIRLEMAN (Cin. '03) from Raleigh, N. C., to Atlanta, Ga., in charge of tuberculosis eradication.

DR. DAVID S. KAY (San Fran. '11) from Seattle, Wash., to South Saint Paul, Minn., on meat inspection.

DR. G. A. POSSE (Colo. '24) from Morehead, Ky., to Mason City, Iowa, on meat inspection.

DR. CURTIS W. BETZOLD (Corn. '32) from Saint Joseph, Mo., to Chicago, Ill., on meat inspection.

DR. E. HEINY (Ind. '08) from Cheyenne, Wyo., to Rocky Ford, Colo., on scabies eradication.

DR. N. G. COBBETT (Colo. '21) from Lamar, Colo., to Las Vegas, N. M., on field inspection.

DR. H. B. FISHBACK (McK. '17) from Fort Atkinson, Wis., to Perry, Iowa, on meat inspection.

DR. C. T. HIGGINBOTHAM (Cin. '18) from South Charleston, W. Va., to Clintwood, Va., on tuberculosis eradication.

DR. CLIFTON CARTER (K. C. V. C. '08) from Salt Lake City, Utah, to Roswell, N. M., on field inspection.

DR. JAMES T. MILLS (K. C. V. C. '16) from Camden, Ark., to Opelousas, La., on tick eradication.

DR. R. C. PATTERSON (Colo. '21) from Emporia, Kan., to Fort Worth, Tex., on tuberculosis eradication.

DR. E. A. GRUBB (Colo. '12) from Olympia, Wash, to Helena, Mont., on field inspection.

DR. EDWARD LAPPLE (Cin. '11) from Sioux City, Iowa, to Saint Paul, Minn., on serum-virus inspection.

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Waldorf-Astoria Hotel, New York, N. Y.
August 13-18, 1934

OFFICERS

Chairman of the Organizing Committee: Dr. A. Eichhorn.
Vice-Chairman: Dr. L. A. Merillat.
Treasurer: Dr. John R. Mohler.
General Secretary (to whom all communications should be addressed):
Dr. H. Preston Hoskins, 221 N. La Salle St., Chicago, Ill.

Membership Campaign

The past month has seen quite a shake-up in the standing of the several states in the campaign for memberships in the Congress. California moved into first place and now leads New York by the scant margin of one member. The Empire State made the greatest numerical gain, having been credited with 33 new memberships during the month. Illinois was forced into third place, Ohio into fourth and Pennsylvania into fifth, as a result of the activities of California and New York. Minnesota appears in the list for the first time, having forced Massachusetts out of sixth place. Likewise, Oklahoma and New Hampshire appear in the standing this month for the first time. Considering the comparatively small number of veterinarians in these two states, their showing is highly commendable. Connecticut, Kansas and New Jersey have been displaced from among the twelve leading states, as the result of the good work done in Minnesota, Oklahoma and New Hampshire. The standing as of April 23:

California	73	Massachusets	36
New York	72	Colorado	35
Illinois	60	District of Columbia	31
Ohio	54	Missouri	26
Pennsylvania	47	Oklahoma	25
Minnesota	37	New Hampshire	20

Sweden and Mexico make their appearance this month among the foreign countries from which applications have been received. Japan still leads, with Canada a close second.

Japan	13	Union of South Africa	7
Canada	12	Sweden	5
Egypt	9	Switzerland	5
Spain	7	Mexico	2

Fourteen other countries have been credited with one member each.

Finances

Two additional contributions have been received from veterinary organizations during the past month. The larger of these was a contribution of $300 made by the Committee on Local Arrangements for the 1933 A. V. M. A. meeting in Chicago. The appropriation was made from a balance remaining in the convention fund after all expenses of the meeting had been paid. This contribution, with one of $200 already credited to the Illinois State Veterinary Medical Association, really makes $500 contributed by Illinois.

Contributions received since the previous listing are:

Committee on Local Arrangements, 1933 A. V. M. A. meeting	$ 300
Nevada State Veterinary Association	25
Previously acknowledged	4,922
	$5,247

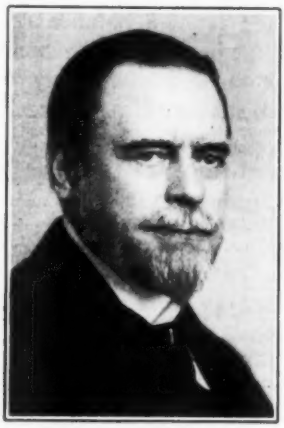
Honor Roll

Twenty-two veterinarians have contributed the sum of $100 each toward the Twelfth International Veterinary Congress, and it is with extreme pleasure that the JOURNAL publishes a list of their names. It is hoped that it will be possible to publish additional lists between now and the opening of the Congress in August. The Organizing Committee believes that it should be possible to secure at least fifty of these $100 contributions, and the members of the Committee are at work with that object in view. Here is the roll to date:

Ackerman, E. B., Huntington, N. Y.
Cotton, W. E., Bethesda, Md.
Crawford, J. Elliott, Far Rockaway, N. Y.
Crawford, J. Stuart, Garden City Park, N. Y.
Eichhorn, A., Pearl River, N. Y.
Goodman, L. W., Great Neck, N. Y.
Hall, Maurice C., Washington, D. C.
Haring, C. M., Berkeley, Calif.
Houck, U. G., Washington, D. C.
Kelser, Lt. Col. R. A., Boston, Mass.
Kock, Herman, Brooklyn, N. Y.
McKim, O. E., Port Chester, N. Y.
MacKellar, R. S., New York, N. Y.
MacKellar, W. M., Washington, D. C.
Marshall, C. J., Philadelphia, Pa.
Millar, Harry C., Asbury Park, N. J.
Mohler, J. R., Washington, D. C.
Noback, C. V., New York, N. Y.
Schoening, Harry W., Washington, D. C.
Schueler, O. R., Brooklyn, N. Y.
Wight, A. E., Washington, D. C.
Zepp, C. P., New York, N. Y.

Doctor Sven Wall on Program

Among the honorary members of the American Veterinary Medical Association who will contribute to the program of the Congress is Dr. Sven Wall, director of the State Bacteriological Institute of Stockholm, Sweden. Dr. Wall has held this position since 1925.



DR. SVEN WALL

Born in Stockholm in 1877, Dr. Wall studied at the Royal Veterinary College in that city and passed the examination as veterinary surgeon in 1901. He served as district veterinary surgeon in Småland, 1902-1904. In the latter year he returned to the Royal Veterinary College as assistant in bacteriology and pathology, a position he held until 1909. Since 1915, he has been a lecturer on the general pathological anatomy of domestic animals at the College. In 1910, Dr. Wall was appointed veterinary surgeon to the Royal Board of Health. From 1912 until 1924, he was veterinary surgeon at the municipal abattoir of Stockholm. It was during this period, in 1917, that he received the degree, Doctor of Veterinary Medicine, from the University of Leipzig.

Dr. Wall has been a voluminous writer and among his contributions to veterinary literature are articles on colic in horses, bovine mastitis, diagnosis of Bang's disease (infectious abortion), and many others. He was a reporter at the Tenth Congress (London, 1914) on the alterations in the uterus in epizoötic abortion and in some other infectious metrites in cows. At the London Congress, he was a reporter in the Section on Meat and

Milk Hygiene. For the past two years he has been editor-in-chief of the *Scandinavian Veterinary Journal of Bacteriology, Pathology and Meat and Milk Hygiene.*

Congress Notes

The first lady member of the Congress to be enrolled was Madame L. Montandon, of Lugano, Switzerland, wife of Dr. L. Montandon, veterinarian in that city.

Dr. E. L. Stubbs, Professor of Veterinary Pathology, has been selected as the official delegate of the School of Veterinary Medicine, University of Pennsylvania.

Dr. A. E. Cameron, Chief Veterinary Inspector of the Department of Agriculture, and Dr. E. A. Watson, Chief of the Pathological Division of the Department of Agriculture, have been appointed government delegates to represent Canada.

Mr. G. H. Locke, M. R. C. V. S., D. V. S. M., of Manchester, England, president-elect of the Royal College of Veterinary Surgeons, will represent that body at the Congress.

Mr. W. Nairn, M. R. C. V. S., of Erichtbank, Blairgowrie, Perthshire, Scotland, will be the official delegate of the National Veterinary Medical Association of Great Britain and Ireland.

Canada Invites Congress Visitors

Those who are planning to attend the Twelfth International Veterinary Congress, in New York City, April 13-18, 1934, are invited to visit Canada, either before or after the Congress. The National Parks of Canada, of the Department of the Interior, at Ottawa, will be pleased to assist in planning attractive tours by supplying necessary information—maps showing the main automobile roads between the United States and Canada, booklets on various phases of recreation in Canada, and a helpful folder entitled "How to Enter Canada." Considerable unpublished data are also available to those making known their specific requirements.

Birthdays Celebrated

Two prominent European scientists celebrated their 70th birthdays recently. On March 18, Professor C. O. Jensen, of Copenhagen, Denmark, was honored by a special session at the Royal Veterinary and Agricultural College, at Copenhagen, attended by veterinarians from Denmark, Norway, Sweden, Finland and Poland. Professor Dr. R. von Ostertag, of Tübingen, Germany, was 70 years old on March 24.

MISCELLANEOUS

Los Angeles County Live Stock Department Celebrates Tenth Anniversary

Members of the Los Angeles County Live Stock Department, Los Angeles, Calif., held a dual celebration on April 4, 1934, that date marking the tenth anniversary of the Department and the twenty-first anniversary of the services of Dr. L. M. Hurt as County Live Stock Inspector.

DR. L. M. HURT

Dr. Hurt outlined the accomplishments of the Department over the ten-year period, stressing the control of important live stock diseases, the activities of the Department in the eradication of bovine tuberculosis and the various services that insure protection to the producers of live stock.

More than 50 prominent dairymen, stockmen and state and county officials attended the function and paid tribute to the work accomplished by the Department. Many of the original sponsors were in attendance and outlined the early history of the County and the live stock problems that necessitated the creation of a well-organized department for handling these problems.

Dr. C. U. Duckworth, assistant director of the California State Department of Agriculture, was a guest and speaker. He spoke especially of his appreciation of the services and valuable coöperative assistance given by the Los Angeles County Live Stock Department.

DR. STANTON YOUNGBERG

Dr. Youngberg Leaves Philippines

Dr. Stanton Youngberg (O. S. U. '07), has severed his connection with the government of the Philippine Islands and has returned to the United States. Accompanied by Mrs. Youngberg, he sailed, on March 8, on the *S. S. General Sherman*, which was due to arrive at San Francisco on April 2. They were scheduled to leave that port on April 5, on the *S. S. Santa Teresa*, for New York City by way of the Panama Canal. Dr. and Mrs. Youngberg were due to arrive in New York on April 23. They plan to visit friends in New York, Philadelphia and Washington and then proceed to Mrs. Youngberg's home in Ohio.

It will be remembered that Dr. Youngberg completed his term of office as Director of the Bureau of Animal Industry, Philippine Department of Agriculture, on December 31, 1932. Since that

time he has been a member of the staff of the Governor General as a technical adviser. Dr. Youngberg had been in the Philippines almost continuously since 1907. He was one of the pioneers in the organization of veterinary sanitary control in the Philippines. It was largely through his efforts that the periodic and deadly outbreaks of rinderpest in the Islands were brought under control. He and his co-workers perfected a vaccine which, in spite of modifications and refinements, is essentially the same as that in use today for the prevention and control of rinderpest.

The *Philippine Herald*, in its issue for January 13, 1934, had this to say of Dr. Youngberg:

> Dr. Stanton Youngberg, who has retired from the government, has been one of the most efficient technical advisers Malacanang has ever had. Upon retiring, he leaves no one possessing his technical knowledge of agriculture and animal husbandry. His valuable services to the government were well recorded in recent letters written to him by Governor General Murphy and Acting Secretary Vargas, of agriculture and commerce.

Dr. Youngberg was also the subject of an interesting and comprehensive biographical sketch in the Oldtimers' Edition of the *Manila Bulletin*, for February 1, 1934.

Horse Comes Back

The horse, it is said, is coming back, but nowhere has he been ushered in so impressively as at Hutchinson, Kan., where horseflesh has just displaced the motorized milk wagon. The dairy owner who, for reasons of economy, turned back the clock to the pre-motor age, made an occasion of it, and the townspeople turned out several hundred strong. The first wagon, shining white and rubber-tired, was ceremoniously christened—with Grade A milk, of course. The wagon-maker, the harness man and the blacksmith made speeches acclaiming the equine renaissance. The cows were among those present and looked on placidly as the barnyard resumed its old precedence over the garage.

This event may mark an epoch of some sort, for all we know. It is a formal recognition that the horse still has a place in a machine civilization, and that he is economical as well. There are certain inducements—no new parts to buy, no battery to run down, no license tags required, no tax on fuel consumed, no hard starting on cold mornings, no new models every year. Hutchinson has plenty to celebrate.

Saint Louis Post-Dispatch.

Colonel Kelser Wins Automobile

Friends of Lt. Col. R. A. Kelser, V. C., U. S. A., of Boston, Mass., who are radio fans were interested to hear an aerial announcement recently, to the effect that Colonel Kelser had won an automobile for a short essay describing the merits of a well-known toothpaste:

LT. COL. R. A. KELSER, V. C., U. S. A.

Colonel Kelser tells just how it was done:

Mrs. Kelser bought a tube of Bost Toothpaste a couple of months ago, and mentioned that she was going to save the carton it came in and submit an answer in an automobile contest sponsored by the company. Nothing was done about it, and once during the following several weeks she threw the carton in the wastebasket but later retrieved it. One Sunday morning, after finishing the newspaper, I saw the carton on the desk, sat down and rattled off a 25-word answer and Mrs. Kelser mailed it in. We both forgot about it, Mrs. Kelser not listening in the night the winner was to be announced, and I being in Washington on my way back from the Ohio State Conference. My father-in-law and a number of my friends in Washington heard the announcement, and called me at the home of Col. Walter Fraser to tell me of it. On investigation, I was assured that my name would not be used in any way, and my winning the car would not be exploited. On that assurance, I accepted a fine Pontiac straight eight.

Nine hundred thirty-nine dollars for twenty-five words. Why not write another book at that rate, Colonel?

12th International Veterinary Congress
New York—August 13-18, 1934

The A. V. M. A. at A Century of Progress

Plans are under way to have the veterinary profession and the activities of veterinarians properly presented to the public at the 1934 World's Fair in Chicago, the second year of A Century of Progress. The management of the exposition very graciously placed a booth at the disposal of the A. V. M. A. early in the year. Several conferences were held with the committee in charge of medical exhibits and the outcome was the assignment of an additional booth, making two in all, with a floor space of 800 square feet, in a very desirable location in the Hall of Science.

The general scheme of the A. V. M. A. exhibit is designed to depict the most important branches of the veterinary profession, or the principal activities of veterinarians, whichever way one cares to put it. So frequently the criticism is heard that the general public has no conception of the width and breadth of the field of veterinary medicine, or the scope of the activities which collectively constitute the work of the modern veterinarian. The exhibit is being designed to present some of these activities, not in the words of a printed page, not in pictures such as are to be found in a college catalog, but by means of three-dimension dioramas as nearly true to real life as human hands can make them.

The present plans call for eight dioramas, each of which will depict an important veterinary activity. It has been rather difficult to select the subject matter, so as to have each branch of the profession represented properly and in a way that would eliminate overlapping or duplication. Some things had to be left out, but this could not be helped. It would take almost double the amount of space available, with a proportional increase in the number of dioramas, to show everything that could be shown in an exhibit of this kind. For example, it would be desirable to feature antemortem inspection of meat animals at abattoirs, testing and inspection of life stock shipped interstate, dipping cattle in connection with tick eradication, tuberculin testing and tuberculosis eradication, poultry practice, etc., but these activities had to be left out. Those to be featured are as follows:

Veterinary education: A veterinary college will be shown in an appropriate setting. A legend will briefly indicate the number of such institutions in the United States, number of students, graduates, etc.

General practice: A farm scene will depict country practice, among all kinds of farm animals. *Preventive medicine* will be emphasized. The cows are accredited tuberculosis-free and Bang's

CERTIFICATE AWARDED A. V. M. A. BY A CENTURY OF PROGRESS

disease-free. The poultry are B. W. D.-free, and the hogs are immune to cholera, etc.

Small-animal practice: The interior of the examination-room of a modern veterinary hospital will be used to present city practice as it is today. The value and importance of *correct diagnosis* will be the dominant idea.

Sanitary science: To portray the *field investigation* of animal diseases, a Texas fever scene will be shown, bringing in the epochal work of Smith, Kilborne and Curtice, insect transmission of infectious diseases, the Panama Canal, etc.

Research work: This diorama will illustrate *laboratory studies* of animal diseases. The interior of a typical research laboratory will be shown and emphasis will be placed on the fact that the animals under observation are not being made to suffer in any way. An effort will be made here to correct some wrong ideas concerning so-called "vivisection."

Food inspection: By directing attention to the inspection of meat and milk by veterinarians, the *public health* phase of veterinary activities will receive the spotlight here.

Military medicine: Here again the inspection of food products of animal origin will be featured, even in greater detail than in the food inspection diorama. This phase of the work of Army veterinarians, including forage inspection, is practically unknown to the lay public, hence the emphasis on it rather than the treatment of sick and wounded animals.

Biological manufacture: The interior of a bleeding-room in a biological manufacturing laboratory will show several horses used in the production of diphtheria antitoxin under veterinary supervision. The legend will bring out the rôle of the veterinarian in this important activity.

On the preceding page is a reproduction of the certificate awarded the A. V. M. A. for the exhibit made at the Fair last year. Those who attended the Chicago convention will remember the exhibit displayed by the Pitman-Moore Company and the Allied Laboratories, Inc., "A Century of Veterinary Progress." This attracted so much attention and received such favorable comment that steps were taken to place the exhibit in the Hall of Science for the balance of the season. The Pitman-Moore officials very graciously turned the exhibit over to the A. V. M. A. and, after many discouraging delays and the unwinding of what seemed like many miles of official red tape, the exhibit, with some slight rearrangement, was installed in the Hall of Science and viewed by several million visitors during the final months of the 1933 Fair.

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The twenty-fourth annual meeting of the State Veterinary Medical Association of Texas was held at the Kyle Hotel, Temple, January 15-16, 1934, with approximately 100 members from all parts of the State in attendance. Several visitors were also welcomed.

The meeting was called to order by Dr. T. O. Scott, of Waco, president. Rev. Michael MarYosip, of Temple, gave the invocation. The address of welcome was given by Mr. W. A. Spencer, of the Temple Chamber of Commerce, and the response was made by Dr. Lewis C. Crabb, of Fort Worth. Dr. T. O. Booth was the very efficient host.

The adoption of a code of ethics and a fair-price schedule, the election of officers and discussions of the papers presented kept the members of the Association busy for the two days of the meeting. Dr. A. C. Burns, of Cleburne, chairman of the Committee on Ethics, presented a code, which was adopted. Reports were made by the following district committeemen: Drs. W. G. Brock, of Dallas; Leon G. Cloud, of Laredo; T. T. Christian, of Waco; W. R. Sanderson, of Brownwood; Frank Lanham, of Amarillo, and Frank Hecker, of Houston.

Papers presented included one on common diseases of sheep prevalent in Texas, by Dr. I. B. Boughton, of Sonora, and one on practical mastitis-control methods, by Dr. James S. Watson, of Mexia. Dr. Hubert Schmidt, of College Station, discussed Dr. Boughton's paper and Dr. Frank Hecker led the discussion on Dr. Watson's paper. Equine encephalomyelitis came in for its share of the attention, with moving pictures of cases and a discussion of the disease being given by Drs. Lewis C. Crabb, of Fort Worth, F. G. Harbaugh, of Lubbock, and Y. J. Aiken and S. C. Ross, of Plainview.

Among the prominent guests attending were: Dr. N. F. Williams, of Fort Worth, past-president of the American Veterinary

Medical Association, who also acted as master of ceremonies at the annual banquet; Dr. H. L. Darby, of Fort Worth; Drs. Ashe Lockhart and C. D. Folse, of Kansas City, Mo.; Dr. H. Wood Ayers, of Oklahoma City, Okla.; Dr. Elmer Lash, of Washington, D. C., and Dr. Ross P. Marsteller, of College Station, Tex.

Officers named to serve during 1934 are: President, Dr. R. L. Rhea, San Antonio; first vice-president, Dr. S. S. Severn, Seguin (reëlected); second vice-president, Dr. Charles W. Neal, San Antonio; secretary-treasurer, Dr. D. Pearce, Leonard (reëlected).

D. PEARCE, *Secretary.*

MISSISSIPPI STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Mississippi State Veterinary Medical Association was held at Greenwood, January 18-19, 1934. The meeting was called to order by Dr. R. H. Stewart, of Jackson, former president of the Association, in the absence of President H. R. Ridgway, of Port Gibson, and Vice-President D. M. Williams, of McComb. The invocation was given by Rev. E. J. Caswell, pastor of the First Baptist Church, of Greenwood, and the address of welcome was made by the Hon. W. K. Clements, of Greenwood. Dr. C. B. Cain, of State College, gave the response.

The business session then followed. Dr. W. L. Gates, of Clarksdale, made a motion to the effect that the Legislative Committee be requested to have the law concerning the appointment of the Board of Veterinary Examiners changed to read as it was before it was amended in 1930. Dr. C. E. O'Neal, of Jackson, reported on the proceedings of the committees appointed by the Extension Service and the Mississippi State Veterinary Medical Association. The revised resolution was adopted by the Association.

The first speaker on the afternoon program was Dr. A. Eichhorn, of Pearl River, N. Y., who gave an interesting and instructive talk on "New Methods of Anthrax Vaccination." Dr. J. W. Giffee, of Lexington, read a well-written paper on "Economic Control of Tuberculosis." A paper, "The Relation of Bovine Tuberculosis and Human Tuberculosis," by Dr. Felix J. Underwood, was read by Dr. H. C. Ricks, of the Mississippi State Board of Health. Dr. S. L. Brister, Jr., of Greenwood, presented a splendid paper on "Tuberculosis in the Human." Dr. C. E. Salsbery, of Kansas City, gave an excellent discussion of "Mastitis."

Dr. H. H. Collins, of Laurel, presided at the banquet in the evening. A number of persons spoke briefly. A delightful musical program included a quartet of negro singers.

Dr. Eichhorn appeared first on the second-day program, his topic being the Twelfth International Veterinary Congress. Dr. Eichhorn stressed the importance of supporting the Congress financially and urged the Association to contribute as much as it could. It was decided that the Association would give $25.

The first paper on the program was given by Dr. J. T. Alston, of Tupelo, and was entitled, "Acetonemia in Cattle," including also a diagnosis for this condition. Dr. W. L. Gates, of Clarksdale, give an interesting talk on "Rabies Vaccination," with a demonstration of how he did community vaccination. Dr. John H. Gillmann, of Memphis, Tenn., discussed some points in small-animal practice. Dr. O. M. Norton, of Greenville, read a paper on "Running Fits in Dogs," which was both interesting and instructive. After the meeting adjourned, a number of members attended a clinic at the hospital of Dr. S. E. Osborne.

Officers elected for the coming year are: President, Dr. A. J. Royal, Scott; vice-president, Dr. N. M. Parker, Jackson; secretary-treasurer, Dr. E. H. Durr, Jackson. The next meeting will be held in Gulfport.

E. H. DURR, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, March 7, 1934, with an attendance of 70.

The speaker of the evening was Dr. Ervin A. Tusak, Jr., the eminent ophthalmological surgeon at Memorial Eye Hospital, and chief of the clinic, Out-Patient Department, Bellevue Hospital, New York City. He discussed, with a contagious enthusiasm for his subject, the anatomical, functional and etiological causes for various disturbances of the eye. Starting with the examination of the eye as a whole, Dr. Tusak went on to discuss the examination of the eyelids, the membrana nictitans and the cornea. He told why we do not have the eye conditions in animals that occur in humans, but said that there are many conditions in animals which resemble those in humans, and that we may expect some interesting developments in animals as work on the eye progresses. So thorough was Dr. Tusak in his discussion that there was little time left to discuss more than a few of the conditions pertaining to the external eye. He has promised to return, however, and give a number of surgical demonstrations. Notice of

this occasion will be given far enough in advance to give out-of-town veterinarians a chance to attend.

April Meeting

The fourth meeting of 1934 was held at the Hotel New Yorker, April 4, with Dr. W. Reid Blair, director of the New York Zoölogical Society, and a past-president of the Association, as guest speaker.

Dr. Blair chose as his subject, "Influence of Disease on Wild Life Cycles," touching on such diseases as tularemia in rabbits, paratyphoid infections and epizoötic encephalitis in foxes, and psittacosis in parrots. The symptoms of these diseases were defined carefully and control measures stressed. Dr. Blair also described the effects of these diseases on humans, discussing symptoms and lesions. In the discussion that followed this interesting paper, both Dr. Blair and Dr. Charles V. Noback, veterinarians to the New York Zoölogical Park, were called upon to answer numerous questions.

At the business session, life membership in the Association was conferred on Dr. George H. Berns, of Brooklyn. Dr. Berns, although retired and confined to his home, keeps up his membership and takes a keen interest in the work of the Association. He is 87 years old.

R. S. MacKellar, Jr., *Secretary.*

OHIO STATE VETERINARY CONFERENCE

A three-day conference for veterinarians was held under the auspices of the College of Veterinary Medicine, Ohio State University, at Columbus, March 21, 22 and 23, 1934. It was the largest conference ever given by the College and the interest of those in attendance was maintained throughout the entire three-day program. It was demonstrated very clearly by the attendance and interest that such conferences are of vital importance to the profession as a whole. Those in attendance received much information that will be of great assistance to them in their respective fields of veterinary service.

The Eighth Annual Veterinary Conference was opened with an address by Dean O. V. Brumley. A cordial welcome was extended to the large number of veterinarians in attendance. Dr. L. W. Goss, chairman of the Department of Pathology of the College, presented the subject, "Pathology of Digestion." He first reviewed the physiology and then the pathology, cleverly sketching his way through the subject on the blackboard.

The next subject on the program was presented by Dr. Richard A. Self, practitioner, of Dallas, Tex., whose subject was a general discussion of hospital business and diseases of dogs and cats. Dr. Self covered many points concerning hospitalization of small animals. He stressed the importance of the proper procedure of admitting an animal to the hospital, complete records, thorough examination and a full understanding of the fees charged.

The last paper on the morning program, "Chemical and Morphological Examination of the Blood and Urine," was presented by Dr. C. E. Hayden, of Cornell University. The physiology and physiological chemistry of blood and urine were reviewed, followed by a discussion of the importance of the various tests made on blood and urine in the diagnosing and treating of diseases. A few tests were made but most of them were left for the afternoon program. Dr. Hayden also gave a simple, inexpensive method for making calcium gluconate solution, which was received with great interest by the veterinarians.

The chairman of the afternoon session, Dr. John N. Shoemaker, again introduced Dr. Self to the assembled veterinarians. This time, he demonstrated mechanical devices used in reducing and casting fractures in dogs. Each appliance was thoroughly described and demonstrated. In fact, a dislocation of the femur at the hip-joint, and a transverse fracture, in dogs, were reduced with these devices. Every one was greatly impressed with the practicability of these appliances.

Dr. C. E. Hayden, assisted by Dr. T. E. Nichols, Ohio State University, demonstrated a number of tests on urine and blood. Of most interest to the practitioners present was the test for acetone bodies. Dr. Nichols' sedimentation test of blood brought forth much discussion.

Dr. W. F. Guard, Director of Clinics, demonstrated bone-plating. He showed a method that is quick, efficient and practical. A number of x-ray photographs were exhibited, and the proper technic of using the x-ray and fluoroscope was discussed.

The first speaker on the Thursday morning program was Dr. J. P. Hutton, of Michigan State College, who presented an able discussion of "The Common Surgical Diseases of the Saddle Horse." The interest shown in this subject demonstrated the fact that the saddle horse is still quite an important factor in veterinary medicine.

Lt. Col. R. A. Kelser, V. C., U. S. A., discussed "Equine Encephalomyelitis." The subject was presented comprehensively and included the history, types, control and treatment. The lec-

ture was illustrated with a motion-picture film showing the different stages and types of the disease.

The third speaker of the morning session was Dr. C. P. Fitch, of the University of Minnesota, who gave a very fine résumé of research as conducted at University Farm. Dr. Fitch supplemented his talk with the showing of a motion-picture film entitled, "Diseases of the Moose."

The last event of the morning session was an address by Mr. George W. Rightmire, president of Ohio State University, who pointed out the value of these conferences to the profession and to the live stock industry. From his remarks, it was evident that he has a keen appreciation of veterinary medicine.

Dr. M. F. Barnes, of the Pennsylvania Bureau of Animal Industry, opened the afternoon program with a consideration of "Bang's Disease and Its Control." Dr. H. G. Bond, of the Ohio Department of Agriculture, led the discussion of this subject, especially as to the control program carried on in Ohio. Both the paper and discussion were well presented and created considerable comment by the audience.

"Some Common Causes of Sterility in Cattle" was the topic very ably discussed by Dr. W. L. Boyd, of the University of Minnesota. This subject brought forth considerable discussion and was one of the features of the conference. Dr. C. E. Hayden gave an interesting paper on "Calcium Metabolism and Therapy." The lecture was supplemented by a demonstration on materials and technic.

The concluding event for the day was an address by Dr. C. P. Fitch, president of the American Veterinary Medical Association, who gave an inspiring talk on the objects of the national association and also discussed the Twelfth International Veterinary Congress.

The third day was devoted entirely to a clinical program, with Dr. W. F. Guard as chairman. The first speaker to be presented was Dr. P. T. Engard, of Marysville, Ohio, who discussed "The Stained-Antigen, Whole-Blood Test for Pullorum Disease." His talk was supplemented by a demonstration of the actual method used in the field. The entire equipment used was carried on a trailer made for this purpose. The method and equipment both showed evidence of considerable thought and originality.

Dr. W. L. Boyd gave an interesting talk on "Sterility of Cattle." A large number of uteri were available, and were used to illustrate many of the conditions encountered in practice. Dr. J. P. Hutton also made his second contribution to the program at this time. His subject, "Tail Operations," was ably discussed and

demonstrations of the various operations common to this region were made. The method of using the various pieces of equipment in conjunction with tail-setting was demonstrated and digital neurectomy was discussed and performed by Dr. Hutton.

Dr. W. F. Guard took part in the program by presenting the subject of "Surgical Treatment of Thoracic Choke." A demonstration of this form of treatment was made and created considerable interest. He also discussed the methods of relieving other forms of choke, particularly cervical choke as produced by small ears of corn or corn cobs.

Dr. W. R. Krill, of the college staff, presented the subject, "Treating for Parasites," and also discussed the desirability of the use of the stomach-tube in the administration of anthelmintics.

The final session was opened by Drs. J. D. Grossman, W. F. Guard and W. R. Krill giving a very interesting presentation of the subject, "Dehorning of Cattle Under Local Anesthesia." Dr. Grossman, by the aid of a previously dissected specimen, demonstrated the location and character of the nerve to be anesthetized. Drs. Guard and Krill made the injection on a living subject, showing the effectiveness of such anesthesia, after which the horns were removed.

Drs. Fitch and Boyd made their third appearance on the program by presenting in their usual forceful manner the subject, "Cyanide Poisoning and Its Treatment" and "Deficiency Disease of Cattle." Both of these subjects were illustrated by motion-pictures.

Dr. D. W. Ashcraft, of the Department of Physiology, demonstrated a "Gad Heart" which consisted of two glass windows mounted in the left auricle and aorta respectively. A bulb syringe was mounted in the left ventricle of the beef heart with an electric light bulb illuminating the interior. By pressing the bulb, water was made to flow through the set-up, demonstrating the action of the bicuspid and aortic valves.

Upon request, Dr. A. B. White, of Grove City, castrated a colt in the standing position.

The final demonstration was the operation on a horse with a fistula of the withers. This was done by Dr. Guard, who used a method of restraint and operative procedure practical for field use.

AMERICAN ANIMAL HOSPITAL ASSOCIATION

As measured by the yardstick of accomplishment, the meeting of the American Animal Hospital Association, held in Cincinnati, Ohio, April 20-21, 1934, was a distinct success. Nearly 50 interested owners of hospitals attended the sessions of the two-day meeting. An encouraging number of members joined the organization at Cincinnati, and more of them gave assurance that their membership applications would be turned in shortly. After the presentation of the report of the Committee on Public Relations, which contained the outline of a plan of action for the organization, this was endorsed by a number of prominent veterinarians.

The program was unique in several respects and obviously was greatly appreciated by those in attendance. On Friday afternoon, from 1:30 until 6:45, an intensive study of the treatment of fractures in dogs and other small animals received the attention of those present. The subject was introduced by Dr. Robert B. Cofield, orthopedic surgeon, Christ Hospital, Cincinnati.

Dr. E. F. Schroeder, Angell Memorial Animal Hospital, Boston, Mass., presented his method of handling fractures, explaining and demonstrating and illustrating with slides as he went along. He held the constant attention of his audience for three hours, during which time be imparted a fund of practical instruction in a masterful style. Dr. Otto Stader, of Geneva, Ill., introduced a new feature in the employment of splints and pins for the reduction of femoral and other fractures.

The session of the second day began with a presentation of "Clinical and Laboratory Studies of the Simultaneous Use of Serum Concentrate (Little) and Living Virus for Immunizing Dogs Against Distemper," by Dr. M. L. Morris, of Stelton, N. J. This splendid paper was discussed by Drs. L. H. LaFond, of Detroit, Mich.; A. R. Theobald, of Cincinnati; Ashe Lockhart, of Kansas City, Mo., and others.

The next meeting will be held probably immediately before the Twelfth International Veterinary Congress in New York in August.

J. V. LACROIX, *Director of Publicity.*

The baboon is the Indian symbol of loyalty, valor and strength: patron of athletes; sponsor of sportsmanship and fair play.

Dhan Gopal Mukerji.

NECROLOGY

CHARLES CHRISTIAN NICKEL

Dr. Charles C. Nickel, of Nowata, Okla., was struck and instantly killed by a passing automobile on the night of January 28, 1934, near Claremore, Okla. Dr. Nickel, surrounded by a small group, was standing near his car which had been damaged but a short time before in a minor accident, when a fast-moving automobile crashed into the group, killing Dr. Nickel and one other man and seriously injuring a third.

Born in Elkhart, Ill., December 26, 1891, Dr. Nickel completed his elementary and high school education in his native city, later entering Valparaiso University. He studied for a time at the Kansas City Veterinary College, but was graduated from the Chicago Veterinary College in 1920. Immediately after his graduation, he established himself in Nowata, where he practiced until his tragic death.

Dr. Nickel joined the A. V. M. A. in 1922. He was also a member of the Oklahoma Veterinary Medical Association, serving as its president in 1932. He was an ardent sportsman, a lover of outdoor life and an adept with gun and rod. He maintained an active membership in the Isaac Walton League. Surviving are his widow (née Thelma Demory), three minor children, five brothers and one sister.

C. H. F.

GERALD RICH

Dr. Gerald Rich died suddenly January 31, 1934, in his adopted home, Augusta, Ga. His death came as a poignant shock to the people of Augusta and to all of us throughout the country who had known and admired him. Dr. Rich was respected as much for his interests in the arts and civic welfare as he was loved for his lively sympathy with the problems of animal conditions.

Born in Philadelphia, Pa., August 13, 1895, Dr. Rich attended the Germantown Academy and Pennsylvania State College before entering the University of Pennsylvania. He received his degree in veterinary medicine from the University in 1920, and was

recognized by his fellow students and professors as one of the finest gentlemen and most promising young veterinarians of the time. Later on, he located at Augusta, where his sterling qualities won for him an intimate place in the hearts of the people.

Dr. Rich joined the A. V. M. A. in 1921. He was a member of Epsilon Chapter of Alpha Psi Fraternity. He is survived by his widow (née Gladys Flower), one son, his mother and one sister.

A. H. C.

WILLIAM M. MILLAR

Dr. William M. Millar, of Portland, Ore., died at the Emanuel Hospital, March 16, 1934, as the result of an automobile accident on March 3. His car was struck by another automobile at a street crossing in Portland as he was going about his official duties.

Born at Ontario, Canada, May 9, 1867, Dr. Millar was graduated from the Ontario Veterinary College in 1907. He had been employed in the Milk Division of the Portland Department of Health for a number of years, and was well known throughout Oregon for his knowledge of tuberculin-testing and the eradication of bovine tuberculosis. Prior to his entering the service of the city, he enjoyed a splendid practice in Portland.

Dr. Miller was a member of the Willamette Valley Veterinary Medical Association and the Pacific Northwest Veterinary Medical Association. He is survived by his widow, one brother and two sisters.

E. E. C.

E. V. DUNBAR

Dr. E. V. Dunbar, of San Antonio, Tex., died March 27, 1934, following an attack of duodenal hemorrhage two days previously. He was 57 years of age.

Born at Halletsville, Mo., Dr. Dunbar moved to Oklahoma when he was 22 years of age, spent one year at the Western Veterinary College, in Kansas City, Mo., and then entered the Saint Joseph Veterinary College, from which institution he was graduated in 1910. Following his graduation, he spent 17 years in general practice at Piedmont, Okla., after which he spent four years in the Rio Grande Valley in Texas. He then located in San Antonio, where he conducted a hospital and general practice until the time of his death.

Dr. Dunbar was a member of the Modern Woodmen for 38 years, an active member of the Oklahoma Veterinary Medical Association during his residence in that state, and a member

of the State Veterinary Medical Association of Texas at the time of his death. He is survived by his widow, three daughters and seven sons, one of whom is Dr. L. A. Dunbar (St. Jos. '20), now practicing at San Antonio.

U. E. M.

ROBERT F. CURRAN

Dr. Robert F. Curran, of Buda, Ill., died in Saint Francis Hospital, Kewanee, Ill., March 28, 1934, following an operation.

Born at Bradford, Ill., March 15, 1889, Dr. Curran grew to manhood on a farm near Lombardville. He was a graduate of the Chicago Veterinary College, class of 1914, and had been in practice at Buda since 1915.

Dr. Curran joined the A. V. M. A. in 1919. He was a member of the Illinois State Veterinary Medical Association, and of the Santa Maria Council, Knights of Columbus, Kewanee. He is survived by his widow (née Mildred Coggins), three sons, five daughters, his father, one brother and four sisters.

HARRY F. DAVIS

Dr. Harry F. Davis, of Arthur, Ill., died at his home, March 29, 1934, as the result of blood poisoning caused by a head injury received while he was treating some cattle a few days previously.

Born at Arthur, July 2, 1882, Dr. Davis attended the local grade and high schools. He then entered the McKillip Veterinary College and was graduated with the class of 1911. Following his graduation, he located at Windsor, Ill., later moving to Iowa. Some years later, he established a practice at Mattoon, Ill., and remained there until about ten years ago, when he moved to Arthur.

Surviving Dr. Davis are his widow (née Jessie Ashworth), three sons, and his father.

JAMES C. McNEIL

Dr. James C. McNeil, of Pittsburgh, Pa., died at his home, April 10, 1934, at the age of 68. At the time of his death he was Superintendent of the Pittsburgh Bureau of Food Inspection, having served in this capacity under ten different mayors.

A native of Pittsburgh, Dr. McNeil was educated in the local schools and then studied veterinary medicine at the University of

Pennsylvania. Following his graduation in 1889, he returned to the Smoky City and entered practice there. In 1895, when the Veterinary Practice Act was amended to provide for a State Board of Veterinary Medical Examiners, Dr. McNeil was appointed by the governor as a member of the first Board, to serve for a term of two years. He received several reappointments to the Board by later governors. For a number of years, and prior to the organization of the Bureau of Food Inspection in 1908, Dr. McNeil was connected with the Department of Public Safety.

Dr. McNeil joined the A. V. M. A. in 1891 and served as a member of the Committee on Incorporation, 1895-1896. He was a member of the Committee on Local Arrangements for both the 1908 and 1927 meetings of the A. V. M. A. in Philadelphia. For many years, "Jimmy," as he was affectionately known by his many friends, took an active part in the affairs of the Pennsylvania State Veterinary Medical Association and was elected to the presidency of the organization in 1905. Although Dr. McNeil was looked upon as one of the wheelhorses of the profession in the Keystone State for many years, he had not kept up his membership in veterinary organizations during recent years. He is survived by his widow (née Edith Smith), one son, one brother and one sister.

FRANK D. KETCHUM

Dr. Frank D. Ketchum, of Pasadena, Calif., died at his home, April 14, 1934. He had been in poor health for the past four years and had been confined to his home for about a year.

Born in Marshall County, Illinois, in 1856, Dr. Ketchum was graduated from the Chicago Veterinary College in 1893. He practiced in Illinois until 1896 when he entered the service of the U. S. Bureau of Animal Industry. His first assignment was at Milwaukee, Wis., on meat inspection. He served as inspector in charge of meat inspection at South Saint Paul, Minn., from 1898 until 1918 when he was transferred to the same position at Wichita, Kan. He remained there until 1927 when he retired from active service, and removed to California.

Dr. Ketchum joined the A. V. M. A. in 1903. He was a member of Braden Lodge No. 168, A. F. and A. M., of Saint Paul, Minn., and Minnesota Consistory No. 1 of the Scottish Rite bodies, also of Saint Paul. He is survived by one sister. His wife preceded him in death a little over a year ago.

F. C. S.

ROY L. RAMSEY

Dr. Roy L. Ramsey, of Lapeer, Mich., died at his home, April 12, 1934, after a protracted illness. He had been a sufferer from paralysis for about three years.

Born at Warsaw, Ind., July 5, 1885, Dr. Ramsey was graduated from the Grand Rapids Veterinary College in 1906. For a number of years he practiced at Mendon and Hillsdale, Mich. Later he was employed by the Michigan Department of Agriculture as county veterinarian in connection with tuberculosis eradication.

Dr. Ramsey joined the A. V. M. A. in 1929. He was a member of the Michigan State Veterinary Medical Association. He is survived by his widow (née Helen Gondre), one daughter, two sons and one brother.

ULYSSES GRANT HOUCK

Dr. U. G. Houck, for 38 years an employé of the U. S. Bureau of Animal Industry, and since 1928 its Associate Chief, died of Hodgkin's disease at his home in Washington, D. C., April 24, 1934, after a brief illness.

Born on a farm in Luzerne County, Pa., January, 3, 1866, Dr. Houck attended local schools and later the State Normal School, at Bloomsburg, Pa. As a youth he was very fond of athletics and taught gymnastics in a local Y. M. C. A. In 1889, he was graduated from Dickinson Seminary with the degree, Bachelor of Science. Several years later, he decided to study veterinary medicine and entered the University of Pennsylvania. He received his veterinary degree from that institution in 1895, and for a period was resident surgeon at the Veterinary Hospital of the University.

In 1896, Dr. Houck entered the service of the U. S. Bureau of Animal Industry as an assistant inspector, and was assigned to meat inspection in Chicago. Later, he was transferred to Sioux City, Iowa, and subsequently was promoted to an inspectorship at Boston. Dr. Houck was a pioneer in the organization of the enlarged meat inspection service provided by the Meat Inspection Act of 1906, and served as Associate Chief of the Inspection Division, from July 1, 1906, until April 15, 1907. Since April 16, 1919, he had been Chief of the Division of Hog Cholera Control. During the 1924 outbreak of foot-and-mouth disease in California, he was in charge of the state and federal forces which finally eradicated this intensely infectious and dangerously treacherous animal plague after it had spread to 16 counties in the State.

Shortly after the Bureau completed the 40th year of its existence, in 1924, Dr. Houck compiled a historical sketch of the accomplishments of the Bureau. This was published in book form, entitled, "The Bureau of Animal Industry of the United States Department of Agriculture, Its Accomplishments, Achievements and Current Activities." It is generally regarded as being the best composite record ever written of the services of the Bureau to the live stock industry and the public.

Dr. Houck joined the A. V. M. A. in 1915. He was a member of the Special Committee on History, 1918-1921, and a member of the Special Committee on the Prevention of Transmissible Dis-

DR. U. G. HOUCK

eases of Animals, 1921-1922. He was a member of the U. S. Live Stock Sanitary Association and had served on the Committee on the Transmissible Diseases of Swine since 1929, for the past two years as chairman; and as a member of the Committee on the Unification of Laws and Regulations, 1925-1928. He was a member of the National Association of B. A. I. Veterinarians.

In his official contacts during his long period of service, Dr. Houck made a host of friends both within and without the veterinary profession. He was affable and courteous under any and all conditions. Few in the government service have had the

variety of assignments that were his during the 38 years of his connection with the Bureau. No matter how difficult or unpleasant the task before him, Dr. Houck always could be depended upon to prosecute it with all his rare tact and indomitable energy. His deep knowledge of veterinary science, particularly sanitary police measures and live stock conditions generally, combined with his administrative ability and good judgment, were widely recognized by control officials everywhere.

PERSONALS

BIRTHS

To Dr. and Mrs. C. W. Picht, of Salt Lake City, Utah, a son, Clyde Wendell, December 2, 1933.

To Dr and Mrs. Greydon Forrest, of Hopkinton, Iowa, a son, John Charles, January 4, 1934.

To Dr. and Mrs. Mack A. Emmerson, of Philadelphia, Pa., a son, Ralph Allen, April 16, 1934.

To Dr. and Mrs. J. Hubley Schall, of Woodside, N. Y., a son, George, April 18, 1934.

PERSONALS

Dr. I. L. Hawkins (Iowa '18) lost his farm home near Cascade, Iowa, by fire on April 16.

Dr. D. G. Quist (Iowa '30), formerly of Ogden, Iowa, is now located in Sioux City, Iowa.

Dr. H. J. Buehler (Mich. '29) has removed from Sloan, Iowa, to Lone Tree, same state.

Dr. H. S. Brundage (Chi. '15) recently opened a new hospital at 5324-26-28 Lake Park, Chicago, Ill.

Dr. William M. Lukens (U. P. '29) has changed locations, from New Hope, Pa., to Harbourton, N. J.

Dr. Myron L. Plumer (U. P. '16), of Newton, N. J., recently purchased a new home at 4 Elm Street.

Dr. George B. Jones (Ont. '94), of Sidell, Ill., was elected supervisor of Sidell Township on March 20, 1934.

Dr. C. H. Stange (Iowa '07) recently completed 25 years as dean of veterinary medicine at Iowa State College.

Dr. L. E. Johnson (O. S. U. '30), of Rushville, Ill., has been confined to his home with an attack of undulant fever.

Dr. R. O. Nye (Iowa '31), of Orion, Ill., has taken over the office and equipment of the late Dr. R. F. Curran, at Buda, Ill.

Dr. Walker France (T. H. '13), of Boonville, Ind., sustained a fractured ankle recently, while treating an equine patient.

Dr. M. J. Huggins (Chi. '13), of Edwardsville, Ill., was a patient at Saint Joseph's Hospital, Highland, the latter part of March.

DR. K. K. GOEKDJIAN (Colo. '30) is operating the hospital of the late Dr. C. W. Eddy, at 3612 Lee Road, Shaker Heights, Cleveland, Ohio.

DR. CHARLES L. COLTON (U. P. '01), for many years a practitioner in Hartford, Conn., now spends about ten months of each year in Florida.

DR. FREDERIC S. JONES (U. P. '08), of the Rockefeller Institute for Medical Research, Princeton, N. J., has been ill with a heart ailment.

DR. W. E. HOOT (St. Jos. '23), of Colesburg, Iowa, and his wife and youngest daughter were painfully injured in an automobile collision on April 15.

DR. F. H. GASOW (Mich. '33), of Mount Clemens, Mich., has opened a hospital for small animals at 144 South Woodward Avenue, Birmingham, Mich.

DR. F. M. KEARNS (Chi. '11), of Louisville, Ky., has been named City Veterinarian. For the past ten years, he has been a captain in the Veterinary Reserve Corps.

DR. CECIL MOULTON (San Fran. '18) has been transferred from San Leandro, Calif., to Sacramento, on meat inspection for the California Department of Agriculture.

DR. J. W. HARKINS (Chi. '14), of Shullsburg, Wis., was narrowly defeated in the race for Mayor of Shullsburg, on April 3, 1934, by Dr. Henry Hosely, a physician.

DR. E. C. JOSS (Chi. '02), Assistant Chief, Meat Inspection Division, U. S. Bureau of Animal Industry, is reported as recovering satisfactorily from his recent illness.

DR. J. H. MURPHY (T. H. '15), addressed the Paris (Ill.) Rotary Club on April 11. His subject was the veterinary profession and its development in the United States.

DR. C. A. FORBES (Chi. '15) has located at Bradford, Ill. He moved there from Henry, where he was county veterinarian for Marshall and Putnam counties for several years.

DR. I. A. MERCHANT (Colo. '24) is on a leave of absence from Iowa State College and has been taking work in public health at the Yale School of Medicine, New Haven, Conn.

DR. A. C. ETCHISON (Chi. '10); of Assumption, Ill., was reëlected chairman of the Republican Central Committee of Christian County, at a meeting held in Taylorville, April 16.

DR. W. W. DIMOCK (Corn. '05), of the University of Kentucky, addressed the Thoroughbred Club of America, at the Phoenix Hotel, Lexington, on February 17. His subject was "Parasites."

DR. L. A. FORGE (Chi. '03), of Burlington, Wis., celebrated his tenth anniversary as Mayor of Burlington, on April 3, 1934, when he was reëlected to that office by an overwhelming majority.

DR. BENJAMIN MCINNES (R. C. V. S. '74), of Charleston, S. C., was Honorary Veterinarian to the seventh annual Charleston Horse Show, held March 14-15, 1934, for the benefit of Pinehaven Sanatorium.

DR. STANLEY PHILIPS (Wash. '34), who finished work for his degree in February, has accepted a position with the Medford (Ore.) Humane Society. He will operate the Society's animal hospital and shelter, and will also engage in private practice.

DR. NEWELL D. BACKUS (Corn. '05) is back in practice again at Elyria, Ohio, after having been in public office for several years. He has just completed a new, fireproof office and hospital for small animals at 345 W. Second Street.

DR. I. D. WILSON, (Iowa '14), of the Virginia Polytechnic Institute, writes that the last county in Virginia has been signed up for tuberculosis eradication and it is expected that the work will be completed some time this year.

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